ARCHIVES OF OTOLARYNGOLOGY

VOLUME 28 OCTOBER 1938 NUMBER 4

COPYRIGHT, 1938, BY THE AMERICAN MEDICAL ASSOCIATION

SUPPURATION OF THE PETROUS PYRAMID WHEN AND HOW TO OPERATE

REPORT OF THIRTY CASES

ROBERT L. MOORHEAD, M.D.

AND

JOHN P. BAKER, M.D.

BROOKLYN

Although sporadic articles on suppuration of the petrous pyramid appeared in the literature previous to 1930, it was not until that year that articles, especially those of Eagleton, Friesner and Kopetsky, focused attention on the subject and established the conditon as a clinical entity. Sufficient time has now elapsed for various observers to report series of cases, and the findings and conclusions in many of these do not correspond with the earlier observations.

This paper is based on a series of 30 consecutive cases in which operation was performed and pus was found in the pyramid. Cases in which infection could not be demonstrated or those in which the patient recovered spontaneously are not included. All but 3 of our patients had had a mastoid operation from a week to several months previously, and in 4 cases petrosal infection followed reinfection of a healed mastoid. In none of these 4 cases were we able to learn about any symptoms of infection of the pyramid at the time of the original operation, although, of course, symptoms may have been present but not noted. It is a well known fact that with an acute condition in the mastoid process there may be symptoms of petrositis—pain in the eye, discharge and positive roentgen evidence, with or without paralysis of the external rectus muscle—without any radical operative intervention being necessary, and on patients who present such symptoms a simple mastoid operation is all that is necessary for a complete cure. Coates, in 1934, reported a series of cases in which there was positive roentgenographic evidence of petrositis with various clinical findings, and in that series the patients made excellent recoveries after simple mastoidectomy. In 1 of our cases (case 18) the simple operation produced only a

Read before the Eastern Section of the American Laryngological, Rhinological and Otological Society, Philadelphia, Jan. 7, 1938.

temporary arrest of the condition, and it was necessary to establish drainage of the pyramid three weeks later.

SYMPTOMS

A few years ago the literature described the pain of petrosal infection as in and behind the eye of the same side, and it was stated that any other pain was not characteristic. Opinion regarding this has changed; so that most observers now agree that, while there may be pain in the eye, pain around the eye or in the frontal region above the eye is just as characteristic and diagnostic if it is present with other symptoms. In this series only 9 patients presented pain in the eye, and, of the others, 1 (case 12) had no pain whatever and 1 (case 18) had pain over the opposite eye. We are unable to explain why the lesion should cause pain on the opposite side. It is simply mentioned as an oddity that was repeatedly observed during the patient's stay in the hospital.

As observed in our patients, the pain is not continuous, sometimes being absent altogether for hours or days only to reappear, and is worse at night, the patients often tossing and moaning in their sleep or waking suddenly with spells of crying.

Pain in the teeth occurred in 4 of our cases. In the first of these (case 2) it was not accorded its true significance, but in the other 3 (cases 5, 22 and 30) its presence aided considerably in the diagnosis. It is our feeling that, together with other signs, pain in the teeth can be just as valuable a diagnostic sign as pain in the eye.

Practically all observers agree that there is a discharge from the middle ear in this condition. In this series there were 6 patients in whom the drum had closed and there was no discharge from the canal. However, the postauricular wound was of course, draining; we do not therefore regard these as real instances of a dry middle ear, for the discharge may have been finding its way out posteriorly. In many cases the discharge was profuse, and in others it was scanty; hence we were unable to observe any real distinction sufficient to be of diagnostic importance. We have not regarded the diminution of the discharge, either slowly or rapidly, as a bad sign unless it is accompanied by an increase of other symptoms, but rather regard a decrease in the amount of discharge as a necessary sequel if the patient is to recover spontaneously.

We have been unable to determine any characteristic temperature. Practically all the patients have some fever. The majority have only a slight rise in temperature, but there are all variations from this to a high septic fever.

Regarding paralysis of the external rectus muscle, opinions have changed. A few years ago one author wrote: "The presence of abducens palsy is more apt to point against than to the diagnosis of petrosal tip suppuration." Of course this paralysis is not a constant finding, but it was present in our series in 14 cases, varying from weak-

ness of the muscle to complete paralysis. We feel that, although its absence does not in any way influence the making of a diagnosis, its presence should be accorded the significance it deserves, and it is definitely of diagnostic value.

We consider roentgen evidence of value only to confirm our clinical diagnosis. It is probably the least valuable and least reliable of all signs in determining what process is taking place in the pyramid and what it is necessary to do for it. Many times films taken in connection with acute mastoiditis show cloudiness of the pyramid without any signs or symptoms accompanying the condition. The cloudiness may be present before any mastoid operation is undertaken, or it may appear subsequent to the cleaning out of the mastoid cells. In cases in which symptoms of petrosal involvement have developed, the roentgenogram may show only generalized clouding without apparent destruction of the cellular partitions.

To repeat, we use roentgenograms only in a confirmatory way, if other symptoms are present, and disregard their evidence if it does not coincide with the clinical diagnosis. We believe that roentgenograms should be taken stereoscopically, with the patient both in the oblique (Stenver) and in the base (Taylor) position.

Transient and usually incomplete facial paralysis, we feel, has not been accorded its true significance. It occurred in 6 cases in this series. We believe that its presence aids materially in the diagnosis.

We consider the time of onset of the various symptoms of as much significance as the signs themselves. Thus, if a patient is operated on for acute mastoiditis, remains well for three or four weeks and then has pain over or in the eye, together with low grade sepsis, the pain is of far greater importance than if it had occurred shortly after the operation. When such signs present themselves and show no diminution after a reasonable period of observation, we feel that in most instances the risk to the patient is decreased if the pyramid is opened.

The character, extent and severity of the pain will in many instances give warning of an advancing process. The patient is more and more restless at night, awakens more often and describes the pain as increasing in severity; there may be considerable pain during the day as well.

If paralysis of the external rectus muscle occurs the time of onset is significant, for paralysis coming on after many days of pain and fever always signifies extension of the suppurative process. This is true also of partial facial paralysis; but the latter may soon disappear, whereas the former invariably persists until the condition is remedied. Repeated roentgenologic examination may show differences in involvement over a period of time, but in our hands this has not been a valuable aid.

Concerning the indication for operation, we must first consider cases of early involvement in which a simple mastoidectomy has not been

done. In these, we feel, except in the presence of meningeal irritation or other urgent signs, a simple mastoid operation should be performed and the result of this awaited. In those cases in which a simple mastoidectomy has been performed, much depends on how recently this has taken place; if the operation was recent and the pain is not increasing, regardless of the increase or decrease in the amount of discharge the patient may be allowed to go along under close observation. The longer after the simple mastoid operation the signs of petrosal involvement appear, the greater the probability that further drainage will be necessary. Thus, if the pain continues to increase, or if paralysis of the sixth nerve makes its appearance at a late date, exploration of the pyramid should be undertaken. If at a considerable period after the simple mastoid operation the pain appears around the eye, accompanied by low grade sepsis, and if this shows no signs of improvement after a few days of observation, it is probably better to explore the pyramid than to wait for meningeal irritation to develop, for there is much less risk in exploration than there is in the meningitis that may suddenly develop.

It is customary with us to make frequent use of spinal puncture, and it is needless to say that any increase in the cell count renders immediate operation more imperative.

CHOICE OF OPERATION

Every surgeon has two objectives in view: (1) to save the life of the patient and (2) to restore function. Every otologist has the duty not only of preserving life but also of conserving hearing so far as this may be possible. Has this duty been respected in the treatment of petrosal suppuration? A review of the current literature on the subject shows that most operators have practically disregarded the hearing. Is this necessary? A review of the statistics on mortality for this condition shows it to be extremely low. In the present series, although 10 patients died 8 of them were operated on in the presence of fulminating meningitis and no other result could logically have been expected. They were operated on simply on the chance that recovery might ensue. In 1 other case (case 2) the condition was unrecognized until too late but in the light of our present knowledge would now undoubtedly be diagnosed and cured.

There is little difference in the mortality in the hands of various operators, and this seems to hold true irrespective of the type of operation performed. On the other hand, comparatively little attention seems to have been paid to the hearing. In 1932 Lillie and Williams recognized the importance of the hearing when they wrote that they had found it "possible in the two cases presented to drain adequately the infected air cells and marrow of the pyramid while conserving the important structures of the middle ear." However, most writers lightly

pass over the question of the hearing with the casual statement that the hearing after petrositis is usually negligible or of little use.

Practically all these opinions regarding the destruction of the hearing are offered by advocates of the radical mastoid operation; hence it is difficult to judge on what ground they formulate their opinions. Naturally, if one performs the radical operation, the hearing is seriously impaired and the ear functionally of little use; but is the radical operation necessary? We do not think so. In this series, only 1 patient had a radical operation, and that was performed before he came under our control.

As the recognition and the treatment of petrosal suppuration have advanced and as the mortality has been reduced to a low figure by practically all operators by various types of operations, we believe the time has now arrived when the otologist should pay more attention to the hearing of the patient. A study of the postoperative audiograms of many of our patients shows that the preservation of hearing can be accomplished if the radical mastoid operation is avoided.

THE CONSERVATIVE OPERATION

We make no claim of originality for our operation. It is a modification of Eagleton's operation of unlocking the petrous pyramid.

Procedure.—Since in practically all cases this condition follows disease of the mastoid, it is assumed that a real exenteration of the mastoid area has been performed, and if it has not it is the first step in the operative procedure. The mastoid incision is extended upward for about 2 inches (5 cm.) and backward from the lower end for about 1 inch (2.5 cm.). After retraction of the periosteum, the removal of bone is commenced and the dura of both the middle and the posterior fossa exposed. The bone between these openings is then removed, so that when the operation is finished the squama has been removed as high up as desired, together with the bone over the sinus and the solid angle between the posterior and the middle fossa, which is the extension of the petrous angle. This solid angle is removed completely down to the labyrinth. The removal of this angle is of double advantage in that it not only allows of much easier elevation of the dura than if the middle fossa alone is opened by a simple decompression, but also permits of tapping of the pontile cistern when the intracranial pressure is elevated on account of meningeal irritation. When this irritation occurs, there is a considerable increase in the amount of cerebrospinal fluid in the basal cisterns, and the withdrawal of this is of advantage both as an aid to the operation and as the first step in combating the spread of the meningitis. This tapping is a routine practice when the cell count of the spinal fluid is above normal.

In the absence of an increased cell count, the intracranial pressure may be reduced by lumbar or cisternal puncture or by tapping of the lateral ventricle. In several instances when the spinal fluid was normal, we found it unnecessary to reduce the pressure in order to reach the pyramid. When the level of the labyrinth is reached, this area is carefully searched for any cellular tracts, and if a tract is found leading into the pyramid drainage is instituted along it and when

possible the tract is uncapped by removing the bone separating it from the middle fossa, as this should afford better drainage than that provided by a small opening. If no tract is found, the dura of the middle fossa is gradually elevated and the anterior surface of the pyramid exposed. In some cases pus is encountered while the surgeon is elevating this area; unless this occurs the surface of the pyramid is inspected and the cortex of the bone opened with a curet or other sharp instrument. In several instances no pus was encountered when a simple opening was made through the cortex, and pus was evacuated only when a search was instituted either inward or backward.

Drainage of the pyramid is established by a strip or two of rubber dam, and the wound is left open.

Advantages and Disadvantages.—The chief objection made is that with this method there is no way to inspect the peritubal area and that if there should be a peritubal fistula it would be missed. This is definitely true, but how often is such a fistula encountered by the surgeon doing the radical operation? The answer varies greatly with different reporters. It is therefore with great difficulty that one can determine the frequency of the fistulas, but certainly they are encountered less often than are cellular tracts in the perilabyrinthine region. Of the latter we had 9 in our series, and the question arises: Should we do a radical operation and destroy the hearing in the rest of our cases simply on the chance that we may find a fistula? It does not seem logical to do so when we can drain the focus, have the patient recover and at the same time have a large percentage of the patients retain good hearing.

The other objections to this operation are theoretic rather than practical. Some surgeons object to the exposure of so much dura. Otologists should be the last to be alarmed by the exposure of the dura of the middle and the posterior fossa. It has long been recognized that in the performance of the simple mastoid operation the exposure of the dura of these regions is without any deleterious effect, irrespective of whether the exposed area is large or small. Objection has been interposed to the removal of a large amount of bone on the ground that it takes away the protection of the brain. This is no longer feared by cranial surgeons. Furthermore, as a large percentage of patients are children, a considerable reformation of bone may be expected, and this has taken place in several instances in the present series.

The objection is raised that the dura is easily torn during elevation, but this is not the experience of surgeons accustomed to work in this location. Objection is also made to the institution of drainage along the surface of a dura which has been scratched or torn, but we have never had a patient in whom meningitis developed or any other ill effects appeared from such drainage. On the other hand, we have had many patients recover who previous to operation had evidence of meningeal irritation, as shown by examination of the spinal fluid and other

tests. We have never had any unfavorable reactions from this operation and regard it as entirely safe and practical. In 1 patient (case 19) drainage of the pyramid was done twice, about two years apart; on each occasion she made a good recovery, and she still has normal hearing. It has been cited as an objection to the elevation of the dura of the middle fossa that the superficial petrosal nerve may be torn, but even if this were so no unfavorable symptoms have been observed as a result.

Thus, most of the objections are didactic rather than practical.

REPORT OF CASES

This series comprises 30 cases, in 20 of which the patient recovered. If, however, we exclude the 8 cases in which operation was performed in the face of fulminating meningitis (in 4 of which—cases 8, 13, 14 and 17—a complete series of tests was not done and a preoperative diagnosis of petrositis was not made), our percentage of recoveries is equal to that attributed to any other method, with the added advantage that a goodly percentage of the patients have serviceable hearing in the ear in which the operation was performed. We have never had a patient in whom the radical mastoid operation could not be avoided.

CASE 1.—A woman aged 40 was admitted to the hospital on Sept. 13, 1930.

History.—The history was that of an acute exacerbation of a chronic condition in the right ear, accompanied by facial paralysis on the right. On September 17 a radical mastoidectomy was performed. After this the patient complained of some headache and was slightly delirious at night; a peritonsillar abscess developed on the right, which was incised, a considerable quantity of pus being liberated. The patient was out of bed and around her room within one week. The next two weeks were uneventful except for an occasional headache. In general she felt well and slept well. On October 12 she had a chill, followed by violent delirium, singing, talking and screaming, and the temperature rose to 104 F. That evening she had her first convulsion. A spinal tap yielded bloody fluid with a somewhat reduced sugar content, but on culture it was sterile. On October 13 she had five convulsions during the day, and the chart showed that she was complaining of headache and severe pain in the right eye. She was semidelirious most of the time. She was seen by one of us at 6 p. m. that day, in consultation.

Examination.—Examination was somewhat unsatisfactory because the patient was mildly delirious and uncooperative. There was some discharge from the ear, and the temperature was 104.8 F. The neck was rigid. Kernig's sign was manifested. Some weakness of the right external rectus muscle was evident. The eyegrounds showed some blurring of the disks, especially on the right side. There was no facial paralysis.

Operation and Later Course.—As the patient seemed to have fulminating meningitis, operation was performed as soon as the operating room could be prepared. The solid angle was removed down to the labyrinth. No fistula into the pyramid could be found. The cistern was tapped and a large quantity of cerebrospinal fluid evacuated. When the dura of the middle fossa was elevated, pus

was encountered just internal to the labyrinth, and an extremely large hole was found in the cortex of the anterior side of the petrous pyramid. Exploration through this wound revealed an enormous cavity, but no attempt was made to curet this. Rubber drains were inserted, and the wound was left open.

On October 14 the entire picture had changed. The patient was much more comfortable; she was rational and was having only moderate headache. On being questioned, however, she stated that she had considerable pain in the right eye. She was eating well and was not vomiting but was slightly delirious that night. From October 15 to October 30, daily spinal taps showed the cell count decreasing from 2,300 to 120. During this time there was no delirium, and the patient was bright and cheerful. There was practically no fever. On October 30 the temperature suddenly rose to 103 F., and the patient was delirious. A spinal tap showed the fluid to be cloudy, and the count revealed 3,000 cells. The sugar content was much diminished. The reaction for globulin was 3 plus. Culture showed Streptococcus mucosus. The wound was reopened, and after the cavity of the pyramid had been gently searched and no further pocketing found, the drains were reinserted. Spinal tap showed the streptococcus to be present on October 31 and November 1, after which the fluid again became sterile. The cell count gradually diminished, and on November 13, the last day that a tap was done, it was 120 and the sugar content was normal. The patient was allowed to get out of bed on November 20.

It was about two weeks after the first exploration of the pyramid before the significance of the history of peritonsillar abscess was recognized. The patient complained of a foul taste in the mouth and said she had lost a tooth. On investigation, an opening was found above and external to the tonsil. A probe could be inserted upward into this opening for over 1½ inches (3.8 cm.), but, as this procedure was accompanied by rather profuse bleeding, no attempt was made to follow it. Fragments of bone were extruded from this fistula at rather frequent intervals for eighteen months. The last fragment that the patient knew of was discharged in June 1931; it caught in the opening and caused so much scratching and discomfort in the mouth that it was snipped loose with scissors by the family physician.

It is of interest to note that roentgenograms of the patient's petrous pyramid taken in December 1934, three years after the operation, show no evidence of the previous suppurative process, and both sides appear approximately alike on the films, despite the extensive necrosis which had taken place.

This case illustrates the formation of an abscess outside of and below the pyramid, which burrowed its way to the pharynx, where it was opened under the mistaken idea that it was a simple peritonsillar abscess. Apparently this procedure afforded sufficient drainage for the time being, but in spite of the pharyngeal drainage the suppurative process in the bone extended, with necrosis and rupture into the middle fossa, and real meningitis developed. This case shows the fallacy of providing drainage only from the inferior surface of the pyramid, as has recently been suggested. Unless the interior of the pyramid itself is drained, the abscess will continue to extend.

Case 2.—A girl of 8 years was admitted to the hospital on Nov. 10, 1930.

History.—The patient had had a mastoid operation on the left ear performed by one of us eight months previously and had been operated on again one

month later because the wound was not healing well. The wound never completely healed, and a small discharging fistula had remained. The discharge from this was intermittent. The ear was dry. For one month prior to the last admission to the hospital the patient had had a low grade fever. At intervals she had had severe pain in the teeth on the left side, so much so that it was necessary to consult a dentist. Three days before admission she first complained of headache. The day prior to admission, the pain in the head had increased and she had vomited on two occasions.

Examination.—The ear was dry. A discharging postauricular sinus was present. The temperature was 104 F. There was severe pain in the head and teeth. The leukocyte count was 49,000 with 87 per cent polymorphonuclears. The spinal fluid showed a count of 3,000 cells, with 60 per cent polymorphonuclears, and no reduction of the sugar content. The neck was rigid. Kernig's sign was manifested. Roentgen examination was not done.

Operation and Later Course.—On November 10 the solid angle was removed down to the labyrinth. The cistern was drained. In front of the vertical canal a small opening was found, which contanied granulations; along the path of this a probe could be inserted for over 1½ inches (3.8 cm.). The fistula was enlarged, and considerable pus escaped under pressure. The pyramid also was opened from the side toward the middle fossa.

In spite of the operation, the meningitis progressed, and the patient died.

At the time this child was seen, the literature on the subject stressed the view that orbital pain was the only pain of consequence and that pain in the teeth was of little diagnostic value. It is now realized that this was an error, and we feel that had the case occurred at the present time the condition would have been recognized and the patient probably saved.

Case 3.—A girl aged 6 years was admitted to the hospital on April 10, 1931.

History.—Six weeks prior to admission the patient had had a mastoid operation on the right. For the last few days there had been pain in the right frontal and right temporal region, but there was no history of pain in the eye. The temperature had had an irregular course. There were no chills.

Examination and Preoperative Course.—The temperature was 102 F. A discharging postauricular wound was present, but there was no discharge from the canal of the ear. There was no rigidity of the neck. The leukocyte count was 10,500. The spinal fluid gave a count of 200 cells. The urine showed pus and casts. The eyegrounds were normal.

Subsequently the temperature returned to normal; the pain disappeared, and the spinal fluid showed a count of only two cells.

On May 11 there was recurrence of the pain in the head, which was confined to the right frontal region, but again there was no pain in the eye. On May 12 the patient vomited, and the spinal fluid at this time showed a count of 2,000 cells. A roentgenogram of the petrous pyramid showed cloudiness with loss of cellular structure as compared with the opposite side.

Operation and Later Course.—On May 15 the solid angle was removed down to the labyrinth. No tract was found. On elevation of the dura of the middle fossa, pus was encountered at a depth of 3 cm. internal to the vertical semicircular canal. The petrous pyramid was opened in this area; more pus was liberated, and a drain was inserted.

Despite the operation the meningitis progressed, and the girl died.

A postmortem examination revealed extensive necrosis of the petrous pyramid with the operative wound opening into this area.

This patient should have been operated on during the first exacerbation of her symptoms, in April. However, as she showed improvement, she was allowed to go along for a month until the second flare-up occurred, with the invasion of the meninges.

CASE 4.—A girl of 13 was admitted to the hospital on Feb. 1, 1932.

History.—On Dec. 19, 1931, she had had an operation for removal of nasal polyps. Two days later she complained of earache and other symptoms. On Jan. 22, 1932, a mastoid operation was done, and this was followed by headache and irregular temperature. On the day of admission the pain in the head became severe; the temperature rose, and the patient vomited. One of us saw her at this time, in consultation.

Examination.—The temperature was 104.8 F. There was stiffness of the neck. Both the middle ear and the mastoid wound were discharging. Kernig's sign was manifested. The patient stated that most of her pain was over the eye. The eyegrounds were normal. The spinal fluid showed a count of 6,000 cells. Roentgen examination of the pyramid showed evidence of destruction of the septums of the cells.

Operation and Later Course.—On the same day, the solid angle was removed down to the labyrinth. A tract of cells found above and posterior to the labyrinth was traced with a curet. The curet dropped into a cavity, and considerable pus welled up. The cistern was drained, but the meningitis progressed, and the patient died on February 7.

For five days following operation, although the patient was perfectly rational and cooperative, she denied having had any pain whatever in the head, but during this time the spinal fluid showed a high cell count, with a steady diminution of the sugar content.

CASE 5.—A man aged 46 was admitted to the hospital on Jan. 30, 1933.

History.—Both ears had been discharging since childhood. The patient had been operated on one year previously by one of us for a temporosphenoid abscess on the left. He had made a good recovery from this operation. For one month preceding admission to the hospital there had been intermittent pain in the right ear, and during the past week this had become severe, radiating to the teeth. The pain in the teeth had prevented him from sleeping for the last three nights. The discharge from the ear had decreased considerably.

Examination.—Examination revealed a discharging ear. The temperature was 100 F. and the leukocyte count 11,000 with 75 per cent polymorphonuclears. A roentgenogram of the petrous pyramid showed definite changes, which were interpreted as indicating destruction. The eyegrounds showed a slight blurring of the left nerve head.

Operation and Later Course.—On February 1, at operation, pus was found in the mastoid. The solid angle was removed down to the labyrinth. No fistula was found, and the surface of the pyramid toward the middle fossa seemed normal. The bone was accordingly opened, whereupon pus was discharged from it.

The pain in the face disappeared in two days. The patient was discharged from the hospital on February 28.

If this case had occurred more recently, it is probable that a more conservative attitude might have been adopted, for the mastoid process had never been opened. It is possible that a simple mastoidectomy might have provided sufficient drainage, although the amount of pus found in the pyramid makes this seem improbable.

CASE 6.—A boy aged 10 years was first seen on June 2, 1933.

History.—The patient had had a mastoid operation on the right on April 9, 1933, and had been submitted to two subsequent operations, the second on May 4.

About May 1, after the boy had experienced considerable pain in the head, paralysis of the right external rectus muscle developed. For four days previous to the time that he was seen there had been pain both in the head and in the right eye. He had also experienced great difficulty in sleeping at night without the use of sedatives and had had a temperature in the evening of about 100 F. There had been four attacks of vomiting in the previous fortnight.

Examination.—The mastoid wound was found to be draining. There was some discharge from the canal. The right external rectus muscle was paralyzed completely with a barely perceptible facial paralysis on the right. There was anesthesia of the right cornea, but the fundi were normal. The leukocyte count was 7,000, with 50 per cent polymorphonuclears. The spinal fluid had a count of 17 cells. There was no rigidity of the neck. There was roentgenologic evidence of extensive necrosis of the petrous pyramid.

Operation and Later Course.—On June 6 the solid angle was removed down to the labyrinth. A tract was found above and posterior to the vertical semicircular canal. A probe could be inserted through this for a distance of over 1 inch (2.5 cm.). When the dura of the middle fossa was elevated, pus was encountered and a necrotic area of bone was found on the anterior surface of the pyramid. The bone was opened through this area into the pyramid. Drains were inserted into this cavity, both from the side toward the middle fossa and through the tract. Culture of material from the cavity showed hemolytic streptococci.

The paralysis of the sixth nerve first showed improvement on June 13, and the patient was discharged from the hospital on July 8.

This patient showed a steady increase in pain, especially at night, and it became more and more localized in the eye. The operation illustrated the risk of relying on drainage of the fistulous tract alone, because had this been done, drainage of the extradural abscess would have been inadequate.

CASE 7.—A boy of 7 years was admitted to the hospital on June 8, 1933.

History.—The patient was transferred from the Contagious Disease Hospital, where he had just recovered from measles. His right mastoid had been operated on for an acute condition on May 9, and from this operation he had made a good recovery. The wound healed, but there was a persistent discharge from the ear. Three days prior to admission his temperature rose, and he complained of pain in the right side of the head and vomited. Twenty-four hours before admission paralysis of the right external rectus muscle was noted.

Examination.—The child appeared acutely ill. He complained of frontal headache but would not admit having any pain in the eye. The temperature was 103.8 F. Complete paralysis of the right external rectus muscle was evident. There was no facial paralysis. The eyegrounds were normal. Slight rigidity of the neck was present. The leukocyte count was 11,000, with 60 per cent polymorphonuclears. The spinal fluid showed a count of 35 cells. The patient vomited several times in the few hours preceding admission. A roentgen examination of the pyramid revealed cloudiness, but no loss of the septums could be demonstrated.

Operation and Later Course.—On June 9 the solid angle was removed down to the labyrinth. No fistula was found. The surface of the pyramid was normal in color, but the bone was soft, and the cavity of the pyramid was easily entered, with liberation of pus. A rubber drain was inserted.

On June 19 the paralysis of the external rectus muscle had almost completely disappeared. The patient was discharged from the hospital on July 8.

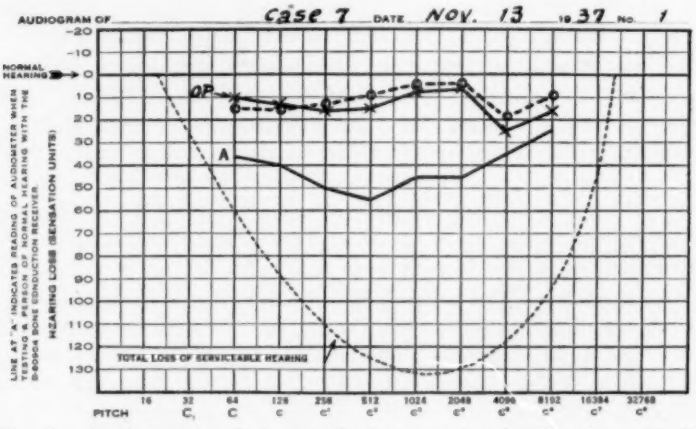

AUDIOGRAM OF Case 7 DATE NOV. 13 1937 NO. 1
NORMAL HEARING
HEARING LOSS (RENSATION UNITS)
LINE AT 'A' INDICATES READING OF AUDIOMETER WHEN PATIENT IS JUST ABLE TO HEAR WITH THE BONE BONE CONDUCTION RECEIVER
BP
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 1.—Audiogram made in case 7 after complete healing, Nov. 13, 1937.

Meningeal irritation was the first sign to attract attention to this patient's condition.

Case 8.—A boy aged 7 years was admitted to the hospital on Feb. 26, 1934.

History.—Five weeks prior to admission he had been operated on for acute mastoiditis. Because of continued high temperature, the wound had been revised. Some rigidity of the neck had been noted five days before, and the onset of vomiting had been noted three days previous to admission. The temperature had averaged 103 F.

Examination.—The temperature was 102 F. There was severe pain in the head. The neck was rigid. Kernig's sign was manifested. The fundi were normal. The spinal fluid gave a count of 10,200 cells, with 75 per cent polymorphonuclears. There was no reduction in the sugar content. A roentgen examination of the petrous pyramid was not made.

Operation and Later Course.—The solid angle was immediately removed down to the labyrinth. A tract of cells was found anterior to the vertical canal, through which a probe could be passed into the pyramid. Pus welled up from this tract. After the cistern had been drained, the pyramid was opened from the side toward the middle fossa.

The meningitis progressed rapidly, and the patient died on February 27.

This patient's petrositis was not diagnosed before operation. He was admitted to the hospital for his meningitis, and the condition was encountered during operative investigation for the meningitis.

CASE 9.—A boy aged 8 years was admitted to the hospital on May 23, 1934.

History.—The patient had had a mastoidectomy on the left two years before. At that time he was said to have had meningitis, with paralysis of the left external rectus muscle. Two weeks before the present admission the left ear became reinfected; the old scar bulged and was incised. Since then there had been frequent frontal headaches, with sharp pain both behind and over the left eye.

Examination.—Examination disclosed a draining postauricular wound. There was a free discharge from the canal. Some weakness of the left external rectus muscle was present. The eyegrounds were normal. The temperature was 103 F. The spinal fluid showed a count of 3,900 cells, with no organisms. Roentgen examination of the pyramid showed cloudiness.

Operation and Later Course.—On May 25 the solid angle was removed. No tract was found. The pyramid was opened. Considerable pus under pressure was found. The cistern was drained and a rubber drain inserted into the pyramid. During the following week the child's condition improved steadily; the temperature dropped to normal, and the cell count of the spinal fluid diminished gradually. On June 7 he was perfectly comfortable and was sitting up in bed and playing. However, on June 8 the pain recurred; the temperature rose to 104 F., and the child vomited. The spinal fluid now showed a count of 8,000 cells. The petrous pyramid was again explored, but no further evidence of retention of pus could be found. The meningitis progressed steadily, and the child died on June 12.

Autopsy revealed purulent leptomeningitis. The petrous pyramid, although necrotic, presented a good opening, and the interior had been well drained.

This case illustrates localized meningitis which at first improved after drainage but then extended and caused the fatal outcome.

CASE 10.—A boy of 8 years was first seen on Oct. 5, 1934.

History.—Three months before, the patient had had a mastoidectomy on the left; the wound did not heal well, and one month later it was revised. Four weeks previously he had commenced to have severe headache, low fever and slight vertigo and photophobia. Ten days previously the pain had become more severe over both eyes, especially the left, and the patient admitted having some pain behind the left eye also. He could not sleep at night except by the use of codeine.

Examination.—Examination revealed the presence of a granulating wound and a profuse discharge from the canal. The left external rectus muscle was weak, but complete paralysis was not demonstrable. The eyegrounds were normal, as also were the visual fields. The blood count showed 9,500 leukocytes, with 55 per cent polymorphonuclears. The spinal fluid gave a count of 2 cells. A roentgenogram of the petrous pyramid indicated the presence of suppuration.

Operation and Later Course.—On October 8 the solid angle was removed down to the labyrinth. No fistula was found. When the dura of the middle fossa internal to the labyrinth was elevated, there was a sudden rush of pus. There was an area of rough bone on the anterior surface of the pyramid, with a small opening into the interior. This opening was enlarged and a rubber drain inserted.

The pain disappeared completely in five days, and the patient was discharged from the hospital on October 30.

This case illustrates that, although there may be necrosis of the cortex of the bone with extradural abscess, the cell count of the spinal fluid does not necessarily indicate this, because in this instance the count was only 2 cells.

CASE 11.—A boy of 6 years was admitted to the hospital on Jan. 5, 1935.

History.—The patient had a double mastoid operation January 5. After this his temperature was irregular; although the blood culture was sterile, both lateral sinuses were exposed, but they appeared normal. There were periods of normal temperature alternating with periods of elevation. About February 15 the patient began to complain of pain in the head, which was limited to the left frontal

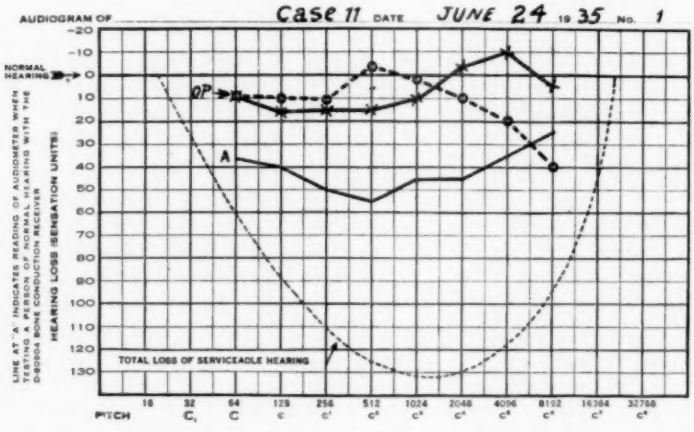


Fig. 2.—Audiogram made in case 11 after complete healing, June 24, 1935.

region and worse at night. There was no complaint of pain in the eye. The temperature gradually rose until it reached about 103 F. each afternoon. On February 20 the boy complained of diplopia.

Examination.—The patient presented low grade sepsis with weakness but not complete paralysis of the left external rectus muscle. The leukocyte count was 18,000, with 60 per cent polymorphonuclears. The count of the spinal fluid was 7 cells. Roentgen examination of the petrous pyramid was inconclusive. However, as the condition was definitely progressing, it was decided to explore the pyramid.

Operation and Later Course.—On February 23 the solid angle was removed down to the labyrinth. No tract was found. The bone on the anterior surface of the pyramid was revealed to be hard. The interior was entered, however, whereupon watery fluid was discharged. A drain was inserted into this opening.

The paralysis of the external rectus muscle was improved, and on the fourth day the temperature quickly returned to normal. The child was discharged from the hospital on March 20.

CASE 12.—A girl aged 13 was admitted to the hospital on Feb. 12, 1935.

History.—On February 12 the patient had a mastoid operation on the right. After this her temperature was about 103 F. every evening. On February 15 paralysis of the right external rectus muscle developed. Her attending physician explored the lateral sinus but found it to be normal in appearance.

Examination.—On February 19 the fundi were hyperemic, but the visual fields were normal. Roentgen examination showed the right petrous pyramid to be cloudy as compared to the left. The spinal fluid revealed a count of 100 cells. There was no pain in the head at any time. The leukocyte count was 9,800 with 82 per cent polymorphonuclears. The blood culture was sterile.

Operation and Later Course.—On February 20 the solid angle was removed and the superior surface of the petrous pyramid exposed. The surface appeared normal, and the bone was extremely hard. On the assumption that the diagnosis was erroneous and that the condition was one of infection of the blood stream rather than of the pyramid, the patient was returned to the ward.

For the ensuing two weeks the temperature in the evening averaged 102 F.; the patient's general condition remained unchanged, and, except for mild postoperative soreness, she had no pain in the head whatever. A spinal tap done on February 23 showed a count of only 20 cells. On March 2 the patient first admitted a slight right or frontal headache with a little pain in the right eye. On March 3 the headache was more marked. The temperature at this time was 104 F. On March 4 the patient had more pain during the day, especially in the top of the head. That evening the temperature rose to 106 F.; she vomited three times and became slightly irrational. Slight rigidity of the neck was now noticed and a spinal tap showed a count of 1,500 cells, with 45 per cent polymorphonuclears. On March 5 it was decided to explore the pyramid. This was exposed and, although the surface was of normal color, slight pressure on the area that had been investigated at the time of the first operation now resulted in an easy entering of the cells. The opening was enlarged and a drain inserted.

The temperature quickly subsided to normal; the paralysis of the external rectus muscle was improved within a week, and the patient was discharged from the hospital on March 22.

This case illustrates the fact that petrositis may exist without pain in any part of the head. The patient was a bright, intelligent girl, and until the latter part of her acute illness she was usually found sitting up in bed chatting with the neighboring patients and apparently suffering no discomfort. For the first three weeks all her symptoms might have been explained on the basis of an infection of the blood stream, and this was thought probable on account of the absence of any pain, even though the blood culture was repeatedly sterile; not until the onset of meningeal irritation did we decide that it was imperative to explore the interior of the pyramid.

CASE 13.—A girl of 6½ years was first seen on March 9, 1935.

History.—Five years previously a left mastoid operation had been done. The wound healed, and the ear became dry. Five weeks before the present admission the patient had had an acute inflammation of the left ear. The scar was incised and a drain inserted. In the preceding nine days she had complained of headache, which was worse at night. No history was elicited of any pain in the

eye. A week prior to admission to the hospital the patient vomited once and not again until the day of admission. The temperature during the week prior to admission had averaged 102 F. at night.

Examination.—Examination disclosed a discharging postauricular wound. There was considerable discharge from the ear. The temperature was 105 F. The neck was stiff. Kernig's sign was demonstrable. The tongue was badly coated. Except for congestion of the veins the fundi were normal. A spinal tap revealed a count of 3,800 cells. No roentgen examination was done.

Operation and Later Course.—The solid angle was immediately removed down to the labyrinth. At this level two tracts of cells were found, one, posterior to the vertical canal, extending only a short distance and not entering the pyramid, and the other, superior to the canal, leading inward, into which a probe could be inserted for about 1¼ inch (3 cm.). When the dura of the middle fossa was elevated, pus welled up, and a necrotic area of bone was found on the pyramid, which was opened through this area. The roof of the superior tract was removed down to this cavity, and rubber drains were inserted.

The meningitis progressed, and the patient died on March 16.

The condition was not diagnosed preoperatively as petrositis, which was found only during operation for the meningitis.

CASE 14.—A boy of 5½ years was admitted to the hospital on March 24, 1935.

History.—A mastoid operation had been done on the right one month previously, and since then there had been periods of elevation of temperature, with pain in the head. In the two days preceding admission the temperature had again become elevated, and there was severe pain in the head, but this was not localized to any particular situation. The patient had vomited once, the morning of the day of admission, and had become somewhat irrational.

Examination.—At the examination the child appeared acutely ill. The mastoid wound was almost healed. There was slight moisture in the canal. The neck was rigid. Kernig's sign was demonstrable. Mentally, the child was not coherent. There was no facial paralysis and no paralysis of the sixth nerve. The blood count was 9,800 leukocytes, with 86 per cent polymorphonuclears. The spinal fluid was cloudy and showed 2,500 cells, with 82 per cent polymorphonuclears. Later the spinal fluid showed hemolytic streptococci. A roentgen examination was not made.

Operation and Later Course.—The solid angle was immediately removed down to the labyrinth. A granulating tract was found anterior to the vertical and above the horizontal canal. A probe passed through this tract entered the pyramid. The roof of this tract was removed, and a large area on the anterior surface of the pyramid was found to be very soft. Rubber drains were inserted.

The child died on March 27.

In this case no diagnosis of petrositis was made preoperatively, and the condition was disclosed only during operation for the meningitis.

CASE 15.—A boy aged 6 years was admitted to the hospital on April 17, 1935.

History.—A mastoid operation was done on the right on April 17. The patient did well until April 25, when the temperature rose to 101 F. and the child complained of pain over the right eye. A scanty discharge continued from the ear. On April 26 the child was apathetic, complained of diplopia and exhibited some

paralysis of the right external rectus muscle. On April 27 he had severe headache, but there was no pain in or behind the eye, only general frontal pain. The discharge from both the canal and the wound had increased markedly. The spinal fluid was bloody. The eyegrounds were normal. Roentgen examination of the petrous pyramid indicated the presence of suppuration.

The record of blood counts was interesting: On April 17 the leukocyte count was 9,200, with 63 per cent polymorphonuclears; on April 19 it was 10,200, with 58 per cent polymorphonuclears; on April 26 it was 16,100, with 55 per cent polymorphonuclears, and on April 27 it was 20,000, with 53 per cent polymorphonuclears.

Because the condition was definitely advancing and the child was rapidly getting worse, operation was decided on.

Operation and Later Course.—On April 27 the solid angle was removed down to the labyrinth. No tract was found. The pyramid was exposed and broken into with a curet, which entered a pocket containing thick yellow pus. A drain was inserted.

The patient made an uneventful recovery and was discharged from the hospital on May 20.

This case offered no difficulty in diagnosis but was one of the few instances in which a study of the leukocyte count was definitely helpful.

Case 16.—A boy of 13 was admitted to the hospital on May 8, 1935.

History.—The patient had had a mastoid operation on the left in 1930 and an incision and drainage of the old mastoid wound two weeks before the present admission. For one week he had complained of frontal headache on the left, with some nausea. He had vomited three days before entering the hospital.

Examination.—The temperature was 100.5 F. Both the left ear and the wound were discharging. The eyes were normal. The leukocyte count was 14,000. The roentgenogram showed some clouding but no destructive changes in the pyramid. Twenty-four hours later the boy complained of pain in the head; he vomited several times; the temperature was 103 F. and the pulse rate 65. There was moderate stiffness of the neck. Kernig's sign was manifested. There was no paralysis of the external rectus muscle. There was definite peripheral facial weakness on the left side. The spinal fluid showed a count of 8,600 cells, with 90 per cent polymorphonuclears.

Operation and Later Course.—On May 9 the solid angle was removed down to the labyrinth. A tract of cells was found anterior to the vertical semicircular canal, but it was impossible to trace this into the pyramid. The cistern was drained and a large amount of cloudy fluid evacuated under great pressure. The pyramid was opened from the middle fossa, with the release of thin watery pus, and a drain was inserted.

Much to our surprise the child showed almost immediate improvement; the meningeal symptoms subsided, and he was discharged from the hospital on June 19.

In the first eighteen hours of this patient's stay in the hospital his condition rapidly grew worse, and, despite this short time for tests it was feared that operation had been delayed too long. The cell count of 8,600 for the spinal fluid is the highest among the cases in our series in which the patient recovered.

Case 17.—A child aged 7½ years was seen in consultation on May 14, 1935.

History.—Nine days previously the child had had a mastoid operation on the right. For the preceding five days there had been headache, with the pain worst over the right eye. The patient had awakened several times at night crying with pain. Four days previously paralysis of the right external rectus muscle had been noted. The child had vomited twice the preceding day.

Examination.—The child was acutely ill. There was a profuse discharge from the canal. The right external rectus muscle was completely paralyzed. There was no facial paralysis. The neck was rigid. Kernig's sign was manifested. The leukocyte count was 16,000. The spinal fluid showed a count of 8,000 cells. No roentgen examination was attempted.

Operation and Later Course.—The solid angle was immediately removed down to the labyrinth. It was very cellular posteriorly, but it was possible to demonstrate a definite tract into the pyramid. The pyramid was explored from the middle fossa. The bone was more grayish than normal and was definitely softened. The cells were entered, and pus was encountered. The cistern was opened, and thick pus escaped, followed by spinal fluid. A rubber drain was inserted into the pyramid.

The child died on May 18.

Although a preoperative diagnosis of petrositis was made, little hope was felt for the child at the time of operation. A complete series of tests was not done, because the child appeared too ill and time was an urgent factor.

Case 18.—A girl 8 years of age was admitted to the hospital on May 11, 1935.

History.—Four weeks previous to admission, after an attack of measles, the patient had had pain and a spontaneous discharge from both ears. That in the right ear had continued. On April 7 paralysis of the right external rectus muscle had developed. She had had an occasional rise in temperature and some general pains in the head.

Examination.—On examination the child did not appear acutely ill, although the temperature was 103 F. The ear showed a pulsating discharge, but no tenderness was present in the mastoid, and there was no sagging of the canal wall. The leukocyte count was 11,000, with 60 per cent polymorphonuclears. The spinal fluid was bloody. The urine revealed the presence of pyelitis. Roentgenograms disclosed some involvement of the right mastoid but a limited petrosal development, some evidence of infiltration but no definite destruction.

First Operation and Later Course.—On May 13 a simple mastoidectomy was done by a competent surgeon. The mastoid was reported to be large and of a pneumatic type, with scattered areas of necrosis. Small areas of both dura and sinus were exposed. During the next two weeks the patient had a low grade fever and complained of pain over the left (opposite) eye, but denied any pain over or around the right eye. The leukocyte count varied from 8,000 to 12,500, being highest on April 16. On May 20 the spinal fluid showed a count of 28 cells. Repeated roentgenograms were inconclusive.

On account of the absence of pain on the right side of the head and the absence of definite findings relative to the tip, together with the presence of pyelitis, exploration of the pyramid was delayed. However, on May 29 the temperature rose to 103 F., the headache was more pronounced and, although there was no rigidity of the neck, there was a slight manifestation of Kernig's sign. On the

morning of May 30 the temperature rose to 104 F.; the child vomited once and presented slight but definite rigidity of the neck. The spinal tap showed a count of 6,400 cells. The patient was immediately sent to the operating room.

Second Operation and Later Course.—The solid angle was removed down to the labyrinth. There was a small area of cells between the floor of the middle fossa and the labyrinth. These were traced with a curet, and the roof of this tract was removed and a gutter to the pyramid thus formed. When the cells of the pyramid were entered, a large quantity of pus welled up. A drain was inserted into this region.

The child made an uneventful recovery and was discharged from the hospital on June 20.

On admission this seemed to be a case in which conservative measures should be tried, because the patient showed definitely acute mastoiditis, and, though there was some involvement of the pyramid, it

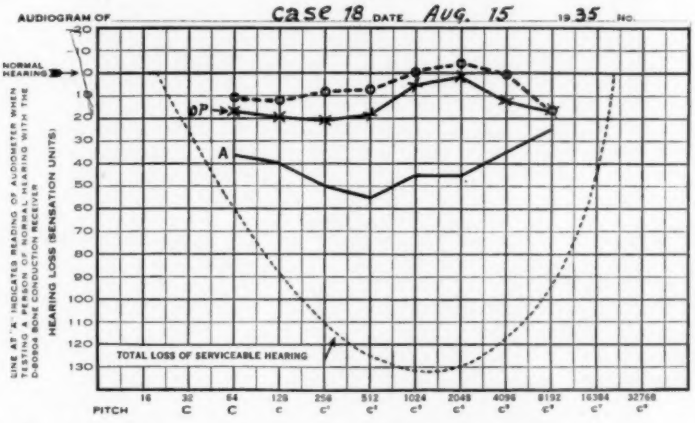


Fig. 3.—Audiogram made in case 18 after complete healing, Aug. 15, 1935.

was felt that cleaning out the mastoid might be all that would be necessary. After the operation the patient mystified us by complaining of pain over the opposite eye. She was repeatedly questioned about this over a period ten days and practically always with the same answer. It was not until meningeal irritation manifested itself that it was decided to open the pyramid. What the factors were that caused the pain on the opposite side we have never been able to determine.

Case 19.—A girl of 13 was seen in consultation on May 28, 1935.

History.—The patient had had a mastoid operation on the right at the age of 10, followed by good healing, and the ear became dry. Three weeks before she was seen earache had developed on the right, followed by spontaneous rupture of the drum. Three days before she was seen the scar had been incised. For the week prior there had been considerable pain in the head and about the right eye, and in the previous four days paralysis of the right external rectus muscle had developed.

Examination.—The child was uncomfortable from pain in the right eye. The temperature was 100 F. The leukocyte count was 15,800, with 74 per cent polymorphonuclears. The spinal fluid showed a count of 8 cells. The eyegrounds were normal. Roentgen examination revealed an enormous petrous pyramid, with a deep layer of cells between the vertical canal and the middle fossa, and definite evidence of involvement of the pyramid.

Operation and Later Course.—On May 30 the solid angle was removed down to the labyrinth. A tract was found above the vertical canal. Following this, a curet entered a large cavity, and a large amount of thick pus escaped. The dura was elevated, and this cavity was entered from above; afterward the tract was partially uncapped. Drains were placed in the cavity both from above and along the tract.

Fig. 4.—Roentgenogram taken in case 19, with the patient in Stenver position and a probe in the petrous pyramid.

The paralysis of the external rectus muscle was improved on June 12. The patient was discharged from the hospital on June 26, and the wound was completely healed about one month later.

The following winter was uneventful, but on Jan. 1, 1937, after an acute infection of the upper part of the respiratory tract, pain developed in the patient's right ear. The drum was found to be bulging and was incised. About ten days later (January 9), the postauricular scar became reddened and swollen, and a simple incision and drainage were done. The wound continued to discharge, and headache developed, especially over the right eye. At this time the drum was closed, and there was no discharge from the canal. The patient soon complained of pain in the right eye. As she still had a temperature up to 100 F.,

she was given an anesthetic, and the old wound was reopened. It was found that there had been considerable regeneration of the mastoid; this was cleaned out, and a tract of cells was found above the labyrinth, but these could not be followed into the pyramid. No trace of the old tract was found. It was decided

Fig. 5.—Roentgenogram taken in case 19, with the patient in the Taylor position and a probe in the petrous pyramid.

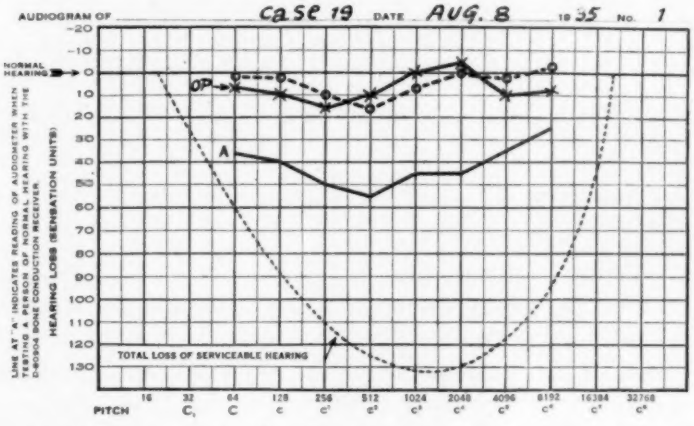


Fig. 6.—Audiogram made after the first operation in case 19 after complete healing, Aug. 8, 1935.

to stop and see what the revision would accomplish, so the patient was returned to bed. After this she was more comfortable. The operative wound drained profusely, and, in spite of the fact that the patient still had occasional pain over the eye, she was sleeping much better and was permitted to return home on February 8.

She was readmitted to the hospital on March 10. At this time the pain in the head and right eye had increased, but there was no history of vertigo, nausea or vomiting. Since February 19 she had again been having a temperature of over 100 F. in the evenings, and for the previous three days she had been drowsy.

On March 11 roentgen examination of the pyramid revealed a localized area of increased density, indicating a destructive process. At this time the blood count showed 11,100 leukocytes, with 66 per cent polymorphonuclears.

On March 15 the patient complained of photophobia and slight nausea.

On March 16 diplopia developed and she vomited her breakfast. The pain in the head now was somewhat more pronounced. Examination of the spinal fluid showed a count of 3 cells.

On March 17 weakness of the external rectus muscle was more marked.

Because the process was definitely advancing, it was decided to reopen the pyramid; therefore on March 18 she was again operated on. A probe was finally inserted into a tract posterior to the labyrinth for a distance of 1½ inch (3.8 cm.); it was left in situ and the patient returned to bed.

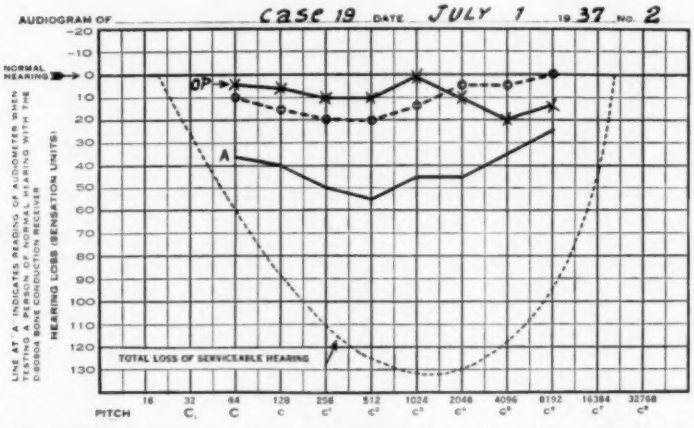

AUDIOGRAM OF Case 19 DATE JULY 1 1937 No. 2
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 7.—Audiogram made after the second operation in case 19 after complete healing, July 1, 1937.

The next two weeks were without any special incident; the pain in the head rapidly disappeared, and the patient was discharged from the hospital on April 2.

This is the only patient we know of who has had two definite attacks of infection of the petrous pyramid separated by a fairly long interval in whom one attack had no relationship to the other. It should be noted that, in spite of two explorations of the pyramid, her hearing is practically normal.

CASE 20.—A girl of 5½ years was seen in consultation on June 28, 1935.

History.—A mastoid operation on the right had been performed on June 15. For one week the patient had had a low grade fever. Three days after her operation she had incomplete facial paralysis on the right, but this cleared up in three or four days. In the twenty-four hours previous to the time the child was first seen her temperature had risen to 102 F., and during the night she had complained of pain in the left eye. She had vomited once the day she was seen.

Examination and Preoperative Course.—Slight drainage from the mastoid wound and a discharge from the canal were revealed. The temperature was 100.5 F. At this time there was no evidence of facial paralysis and no rigidity of the neck.

Hospitalization was advised, but an uncle of the patient, a physician, refused to have her moved, because in his opinion her condition was improving.

On the morning of June 30 the temperature rose to 103 F.; the pain in the head increased; the child vomited; rigidity of the neck made its appearance, and a spinal tap showed a count of 5,400 cells.

Operation and Later Course.—The solid angle was immediately removed down to the labyrinth. No tract was found. The pyramid appeared normal on the surface, but when an opening was made into the bone considerable pus welled up. A drain was inserted to this area after the cistern had been drained.

In spite of the operation, the meningitis progressed, and the girl died on July 11.

This patient should have been operated on and should have recovered, but such an outcome was prevented by meddlesome interference. The pain in the eye, the temperature and the transient facial paralysis were sufficient to establish the diagnosis, and the increasing pain and rising temperature showed that it was not a subsiding process.

Case 21.—A girl aged 8 years was admitted to the hospital on Feb. 26, 1936.

History.—The child had been seized with an earache on the left on January 15. Spontaneous opening of the drum had occurred on January 19. Since then the ear had been discharging, and the patient had been having an irregular temperature. There was considerable pain in the head and over the left eye and also some pain in the eye. There had been no vomiting. The child had not had any chills. She was restless at night. Diplopia developed the day before admission.

Examination.—Examination revealed a discharging left ear. The temperature was 99.5 F. The blood count showed 9,800 leukocytes, with 63 per cent polymorphonuclears. The spinal fluid had a count of 5 cells. Paralysis of the left external rectus muscle was present. The roentgen examination definitely indicated petrositis.

Operation and Later Course.—On February 27 the solid angle was removed down to the labyrinth. No fistula was found. No pus was encountered on the surface. The bone was of normal color. The pyramid was opened, and thin watery pus escaped. A drain was inserted, and the patient was returned to bed.

On March 2 improvement was first noted in the paralysis of the external rectus muscle, and from that time on the child continued to improve and went on to an uneventful recovery. She was discharged from the hospital on March 15.

In this case it was debated whether to do a simple mastoid operation and await results or to drain the pyramid. The child appeared sicker than one would ordinarily expect from simple mastoiditis. The pain was severe. This, coupled with the late appearance of paralysis of the external rectus muscle and roentgenogram apparently showing definite destruction in the pyramid, prompted us to proceed with drainage of the pyramid.

CASE 22.—A girl of 13, was admitted to the hospital on March 16, 1936.

History.—In 1932 the child had had a simple mastoid operation on the right, from which she had completely recovered. On Feb. 24, 1936, pain in the right ear appeared, with spontaneous perforation of the drum. In a few days the scar became reddened and bulging; it was therefore incised and a drain inserted. At this time there was pain in the right lower jaw and teeth. In spite of drainage, pain and fever persisted; hence on March 17 a complete revision of the mastoid was done, as there had been considerable regeneration of bone since the original operation in 1932.

The pain in the head and in the right lower teeth continued; the temperature was irregularly elevated, once reaching as high as 105 F.; there was rather profuse perspiration; the leukocyte count was 22,000, with 86 per cent polymorphonuclears; a spinal tap on March 21 revealed a count of 180 cells, but there was no rigidity of the neck. A roentgenogram of the petrous pyramid showed it to be cloudy, but no definite destructive changes were noted. On March 21 the patient vomited twice; it was therefore decided to open the pyramid.

Operation and Later Course.—On March 22 the solid angle was removed down to the labyrinth. No fistula was found. When the dura of the middle fossa was elevated beyond the labyrinth, thick yellow pus was encountered between the dura and the bone. No opening into the bone could be demonstrated; the pyramid was therefore opened with a curet, and more pus was evacuated. Rubber drains were inserted.

After operation the pain in the teeth disappeared, but the headache and fever persisted for some time. On March 27, at the time of the first dressing, the opening in the pyramid was inspected and found to be draining satisfactorily, and the drain was reinserted.

The patient gradually improved and was discharged from the hospital on April 20.

This patient did not present the usual picture of a pyramidal involvement, and operation was therefore delayed until the signs of meningeal irritation developed.

CASE 23.—A boy 12 years of age was admitted to the hospital on April 6, 1936.

History.—The patient had had a mastoid operation on the left on March 13 and one on the right on March 17. After these he had made an apparently good recovery, and the temperature had reached normal on March 25. He had first complained of pain in the right eye on March 18, the day after the mastoid operation on the right, and had continued to do so at irregular intervals. He perspired freely; the tongue was coated; the temperature was irregular, reaching as high as 102 F., and he was frequently restless at night because of the pain in the head and the right eye. There was occasional nausea but no vomiting.

Examination.—On admission, examination revealed the discharge from the right ear persisting. The postauricular wound was not completely healed. The temperature was 100 F. The blood count showed 10,300 leukocytes, with 83 per cent polymorphonuclears. The spinal fluid gave a count of 35 cells. A roentgenogram of the petrous pyramids was suggestive of involvement but was not absolutely definite. On April 7 the patient complained of pain in the right eye and the right lower teeth; the temperature rose to 103 F., and he was nauseated. At this time the spinal fluid showed a count of 60 cells.

Operation and Later Course.—On April 7 the solid angle was removed down to the labyrinth. No fistula was found. The surface of the pyramid was normal, but when an opening was made into the pyramid greenish yellow pus welled up under considerable pressure, and a pronounced foul odor was noticeable. Culture of the pus showed hemolytic streptococci.

After the operation the convalescence was uneventful, and the patient was discharged from the hospital on May 9.

This child was admitted with definite petrositis, and during the twenty-four hours necessary to complete the examinations his condition definitely advanced.

CASE 24.—A man of 58 was admitted to the hospital on April 20, 1936.

History.—A mastoid operation on the right had been done in a southern city on March 24, on account of severe temporal pain occurring with a mild infection of the middle ear. After the operation the temporal pain had continued,

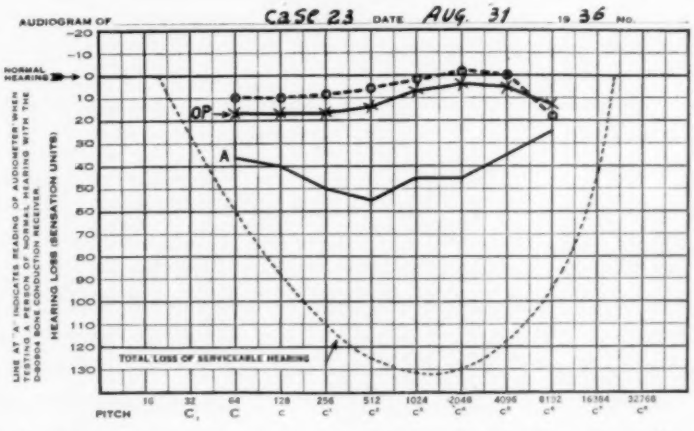


Fig. 8.—Audiogram made in case 23 after complete healing, Aug. 31, 1936.

and one week prior to admission to the hospital the patient had noticed blurring of vision of the right eye. A spinal tap at this time had showed a count of 5 cells, and the temperature had been almost normal. Five days before admission paralysis of the right external rectus muscle had first been noticed. The patient stated that the pain was severe at night; it began in the right frontal region and radiated backward. There was no pain in the eye or the teeth. There was occasional nausea but no vomiting. Because of the diplopia, the patient kept his right eye closed.

Examination.—Examination revealed the postauricular wound to be draining. There was a moderate pulsating discharge from the middle ear. The right external rectus muscle was weak but not completely paralyzed. No facial paralysis was present, and no muscular incoordination was demonstrable. The fundi were normal. A roentgen examination of the petrous pyramid revealed the right side to be cloudy. There was marked cellulation, but the cells were not clearly outlined as compared to those on the left side. The blood count showed 28,200 leukocytes, with 80 per cent polymorphonuclears. No spinal tap was done.

Operation and Later Course.—On April 21 the solid angle was removed down to the labyrinth. Profuse bleeding and oozing were present in the whole operative area. The dura of the middle fossa was raised and the petrous pyramid exposed. However, the surface of the bone appeared normal. The bone was opened, but no free pus could be demonstrated because of the bleeding, which obscured accurate observation. However, the interior of the pyramid was definitely softened, and a rubber drain was inserted into the bone.

After the operation the patient had increased pain. The next day the frontal headache continued; the patient was slightly irrational, and late that afternoon he had a slight chill. On April 23 he became rather stuporous but was definitely delirious when aroused, and the temperature rose to 104 F. No signs of defects of the cranial nerves, except of the sixth, were noted. There was no rigidity of the neck, and Babinski's sign was not manifested. There was no slowing of the pulse. The patient quickly became comatose, but still there were no signs of localizing import. The laboratory reported the blood culture positive for Str. mucosus. The coma continued, and the patient died on April 25.

Autopsy showed the petrous pyramid to be necrotic, with a satisfactory opening for drainage on the anterior surface. There was also a large superficial abscess situated over the right parietofrontal region, which undoubtedly was the cause of death, but which, so far as could be determined, had never caused any localizing signs. No relationship could be established between this abscess and the necrosis of the petrous pyramid.

This case is interesting in that it apparently shows two unrelated processes arising from the ear. The enormous amount of bleeding during the operation certainly was due to the frontal abscess. Although the intracranial pressure did not seem to be greatly increased and there was no necessity for the withdrawal of any spinal fluid, it seems probable that a large part of the pain from which the patient suffered was due to the abscess, in spite of the fact that he had advanced petrositis, as shown by the late appearance of paralysis of the external rectus muscle.

Case 25.—A girl of 8 years was admitted to the hospital on March 3, 1937.

History.—Three weeks before admission the patient had had pain in the right ear, followed by spontaneous perforation of the drum. Since then she had complained of some frontal headache, and her temperature had been following an irregular course, reaching as high as 101 F. Two weeks before admission to the hospital her father noticed that her eyes converged. She vomited once, the day before she entered the hospital.

Examination.—A discharging right ear, a sagging canal roof, a temperature of 100 F. and a pulse rate of 100 were revealed. There was no paralysis of the right external rectus muscle, no vertigo and no nystagmus. The blood count showed 15,000 leukocytes, with 75 per cent polymorphonuclears. The spinal fluid was bloody. The roentgenogram showed considerable involvement of the mastoid, but no definite changes were noted in the petrous pyramid. On March 6 a simple mastoid operation was done, disclosing a large cellular mastoid.

There was the usual postoperative reaction until March 9, when the temperature rose to 102 F. and the pulse rate to 120. The patient complained of some headache, especially during the night, but it was not localized to any one particular

region. The cervical glands became swollen and tender. On March 10 the patient vomited, and the temperature became elevated to 104 F., with a pulse rate of 134. A spinal tap at this time revealed a count of 1,200 cells, with 50 per cent polymorphonuclears, and a normal sugar content; it was later reported as sterile. There had been no change in the paralysis of the external rectus muscle.

Operation and Later Course.—On March 10 the solid angle was removed down to the labyrinth. No fistula was found. The cistern was drained. The petrous area was exposed. The bone appeared normal on the surface. The pyramid was opened, whereupon pus was evacuated under pressure. A rubber drain was inserted.

On March 17 some improvement was first noted in the function of the external rectus muscle. The patient was discharged from the hospital on April 23, 1937.

This case illustrates involvement of the pyramid at the time of the invasion of the middle ear. However, the simple mastoid operation was not sufficient to establish drainage, and meningeal irritation soon

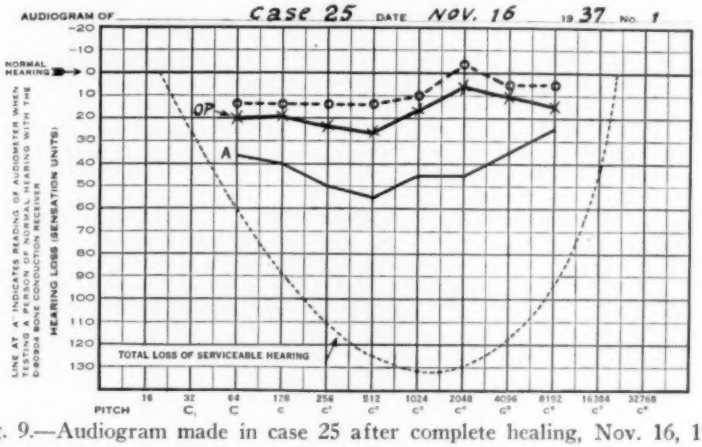


Fig. 9.—Audiogram made in case 25 after complete healing, Nov. 16, 1937.

developed. There were no signs before that time which pointed to the necessity for operation at an earlier date.

Case 26.—A boy of 10 years was admitted to the hospital on March 5, 1937.

History.—There was a history of a mastoid operation on the left on January 26, followed by an uneventful recovery until the end of the second week. At that time the temperature rose to 102 F., and the patient complained of severe headache and photophobia. For the next week there was intermittent temporal pain and some pain in the left eye, the discharge from the ear and from the wound increased and the patient had a low grade fever. For two weeks he was free from symptoms. About March 1 the pain recurred, being especially severe at night and localized in and about the eye. Weakness of the left external rectus muscle also appeared, and the child was readmitted to the hospital.

Examination.—A discharge, both from the aural canal and from the postauricular wound was revealed. The temperature was 101 F., and the boy was complaining of pain in the left eye and the left frontal region. Incomplete paralysis of the left external rectus muscle was present. The fundi were normal. The leukocyte count was 15,300 with 82 per cent polymorphonuclears. The spinal

fluid was bloody. The roentgenogram showed the cellular structure of the pyramid to be cloudy and indistinct, with questionable evidence of a break in the roof near the pyramid.

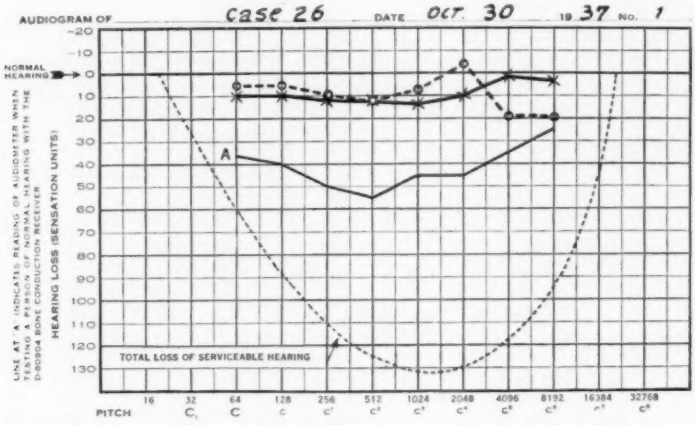

AUDIOGRAM OF case 26 DATE Oct. 30 19 37 No. 1
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
A
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 10.—Audiogram made in case 26 after complete healing, Oct. 30, 1937.

Fig. 11.—Roentgenogram taken in case 27 with the patient in the Stenver position and a probe in the petrous pyramid.

Operation and Later Course.—On March 9 the solid angle was removed. No fistula was found. The dura of the middle fossa was elevated, and the pyramid appeared normal. The bone was entered, and foul-smelling greenish pus was evacuated. Culture of this showed hemolytic streptococci.

By March 12 the weakness of the external rectus muscle could no longer be demonstrated; recovery was rapid, and the child was discharged from the hospital on April 3.

Fig. 12.—Roentgenogram taken in case 27 with the patient in the Taylor position and a probe in the petrous pyramid.

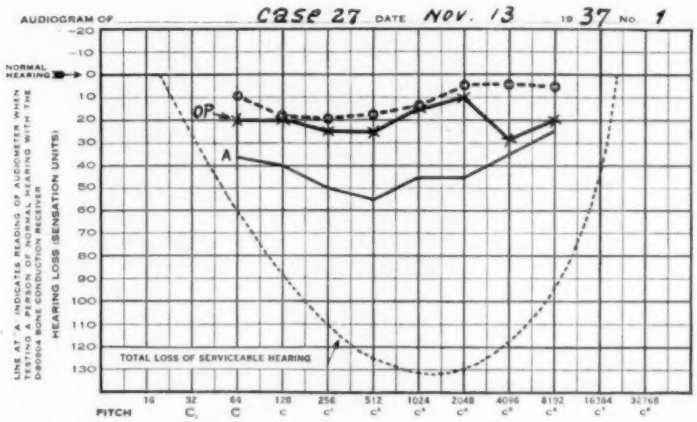


Fig. 13.—Audiogram made in case 27 after complete healing, Nov. 13, 1937.

This child was observed over a period of almost five weeks, and at the end of that time it could be easily seen that the process was not subsiding. Operation confirmed this.

Case 27.—A boy of 5 years was admitted to the hospital on April 13, 1937.

History.—Four weeks before entering the hospital the patient had had a mastoid operation on the right. This was followed by the usual course until one

week prior to admission, when the boy complained of frontal headache on the right. He vomited in the twenty-four hours antedating admission, and his vomiting was accompanied by a rise of temperature.

Examination.—No discharge from the aural canal was revealed but there was some discharge from the postauricular wound. The temperature was 104 F. and the pulse rate 120. No facial paralysis and no paralysis of the sixth nerve was present. There was no nystagmus. The neck was slightly rigid. The spinal fluid was cloudy and gave a count of 5,000 cells, with 80 per cent polymorphonuclears. The sugar content was normal (80 mg.), and the fluid was later reported as sterile. The blood count showed 14,800 leukocytes with 76 per cent polymorphonuclears. The roentgenogram of the petrous pyramids showed the right to have increased density throughout, but no definite changes beyond that could be made out.

Operation and Later Course.—On April 13 an operation was performed. There were cells and granulations extending inward behind the labyrinth for a considerable

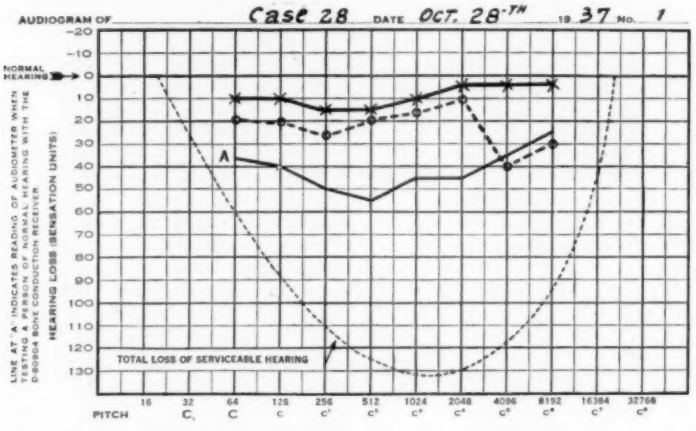

AUDIOGRAM OF Case 28 DATE Oct. 28TH 1937 No. 1
NORMAL HEARING
HEARING LOSS (SENSATION UNITS)
LINE AT 'A' INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE D-90904 BONE CONDUCTION RECEIVER
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Fig. 14.—Audiogram made in case 28 after complete healing, Oct. 28, 1937.

distance, but no tract could be found extending to the pyramid. The cistern was drained. The surface of the pyramid was normal in appearance. The bone was broken open with a curet, and pus was obtained on searching toward the apex. A rubber drain was inserted.

The patient was discharged from the hospital on May 18, 1937.

This child definitely had meningeal irritation on admission to the hospital; he was therefore operated on at once.

CASE 28.—A boy of 7 years was first seen on June 4, 1937.

History.—A mastoid operation on the left had been done on February 7, followed on March 4 by opening and draining of the lateral sinus because of sepsis. The patient had been discharged from the hospital on March 31. During that time there had been no symptoms suggestive of involvement of the petrous pyramid. The wound never healed completely, and for the ten days preceding the time when he was first seen the child had been complaining of severe frontal headache, more marked over the left eye. The pain in the head had awakened him several

times each night, and on such occasions he would cry out with pain. Two days before one of us saw the patient he had had several attacks of vomiting, and on the same day diplopia had been noted.

Examination and Preoperative Course.—The child was apathetic, complaining of photophobia and wanting to be left alone. There was a discharge from the aural canal and from the postauricular wound. The temperature was elevated to 101 F. Complete paralysis of the left external rectus muscle was noted. The fundi were normal. There was no facial paralysis. The roentgenogram showed considerable involvement of the left pyramid.

Operation was advised, but, as the family did not accept the advice kindly, it was delayed. In a few days the child improved, so that he was having only an

Fig. 15.—Roentgenogram made in case 29 with the patient in the Stenver position and a probe in the petrous pyramid.

occasional headache. He remained comfortable, with no change in symptoms, until June 20, when he again had an attack of vomiting. That night there was a recurrence of the severe headache, with suspicious rigidity of the neck, and the spinal fluid revealed a count of 3,000 cells. Operation was now imperative.

Operation and Later Course.—On June 21 the solid angle was removed down to the labyrinth. A tract was found anterior to the vertical canal, which contained granulations but no free pus and did not seem to open well into the pyramid. The cistern was drained. The dura of the middle fossa was elevated, and an extradural abscess was encountered. The bone was then opened and more pus evacuated. A rubber drain was inserted into the pyramid.

The child made an uneventful recovery and was discharged from the hospital on July 14.

There probably would have been much less risk if this child had been operated on two weeks earlier, as there was evidence at that time of meningeal irritation, but because this was of short duration it was necessary to delay operative intervention.

CASE 29.—A girl of 11 years was seen in consultation on Oct. 11, 1937.

History.—The history obtained was that the child had had a mastoid operation on the left on September 9, after an acute condition lasting two and a half weeks. The only prominent feature in the history was severe headache, which had commenced at the time of the acute infection of the middle ear. After the operation the child had made a good recovery, but on October 5 the headache had reappeared and had become localized over the left eye.

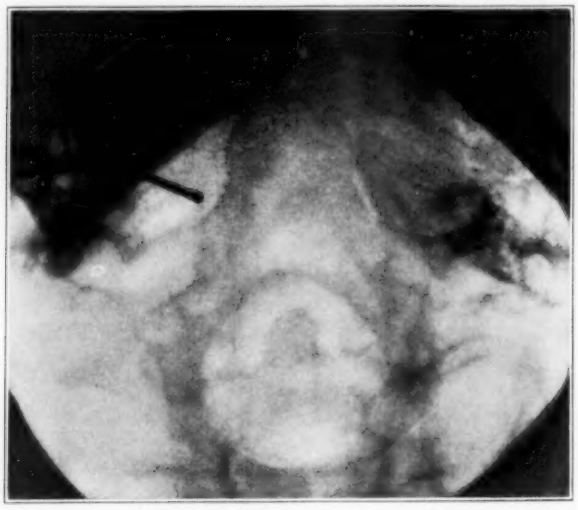


Fig. 16.—Roentgenogram made in case 29 with the patient in the Taylor position and a probe in the petrous pyramid.

Examination.—The girl was bright and alert; she complained of pain in the left frontal region but, on questioning, said she had some pain in the eye itself. The temperature was 101 F. and the pulse rate 120. There was no discharge from the external ear and only a slight discharge from an almost healed operative wound. There was no facial paralysis or paralysis of the external rectus muscle. The blood count showed 12,500 leukocytes, with 68 per cent polymorphonuclears. The spinal fluid gave a count of 57 cells, mostly polymorphonuclears. A roentgenogram of the petrous pyramid disclosed definite clouding, with a probability of destructive changes.

Operation and Later Course.—On October 12 the solid angle was removed. No fistula was found. The cistern was drained and the dura of the middle fossa elevated, exposing the pyramid. The bone was easily entered with a curet. No

pus was encountered on entrance, but exploration of the interior of the pyramid resulted in the welling up of pus into the wound. A rubber drain was inserted into the cavity of the pyramid.

The child was discharged from the hospital on November 10.

CASE 30.—A woman aged 27 was first seen on Oct. 15, 1937.

History.—In March a double postauricular mastoidectomy had been performed. Both wounds had healed in six weeks, but since then the right ear had always felt full. Both ears were dry all summer. On September 25 the patient had had an attack of headache, vertigo and vomiting. She was seen at this time by a competent otologist, but except for mystagmus to the left there were no neurologic signs. Two days later the right drum was incised, and pus was obtained. The ear had been discharging since that time, and there had been considerable pain in the head, more on the right side, frontal, over the eye and occipital. She had had to take considerable acetylsalicylic acid at night to sleep. She had had no fever, and the pulse rate had been normal.

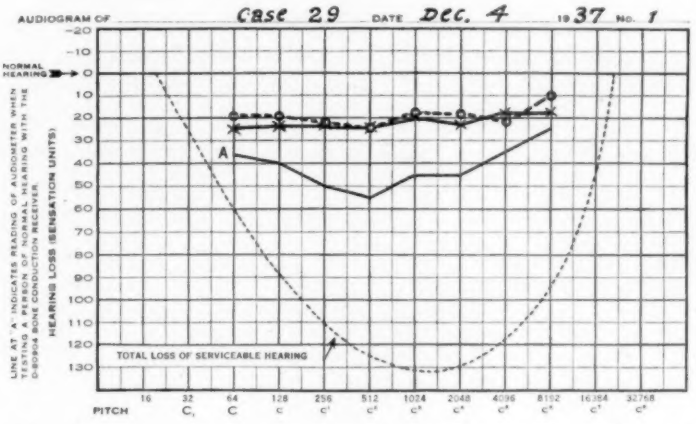


Fig. 17.—Audiogram made in case 29 after complete healing, Dec. 4, 1937.

Examination and Previous Operation.—The right ear showed a pulsating discharge; the drum was thickened; there was a definite sag to the canal roof; the mastoid was not tender; there was no fever, and the pulse rate was normal. The right ear was absolutely deaf, and there was no reaction of the labyrinth. There was spontaneous nystagmus to the left, but the pupils and eyegrounds were normal. Roentgen examination of the petrous pyramids at this time showed changes that were interpreted as indicating a destructive process. Although involvement of the pyramid was suspected at this time, a simple mastoid operation was done. The mastoid was found to be large and pneumatic, with considerable pus in the cells. She made an uneventful recovery from the operation and left the hospital at the end of one week. The middle ear was dry and the drum closed at the end of two weeks. The discharge from the postauricular wound gradually lessened but never completely stopped. For the seven weeks following the mastoid operation the patient had some irregular pain in the right side of the head and over the eye, and she also complained of some pain in the teeth. On December 14 the pain increased, and she was awake most of the night with pain in the right side of the face and teeth, with some pain over

the right eye. There was no facial paralysis, but the face was not symmetric (causing involvement of the facial nerve to be suspected). The right pupil was occasionally larger than the left, and the right palpebral fissure was also larger. The eyegrounds were normal. There was no paralysis of the sixth nerve. The temperature was said to be normal. Roentgen examination showed that the changes in the pyramid had increased as compared to those shown in roentgenograms taken eight weeks before. A spinal tap just before the operation showed 4 cells per cubic millimeter.

Operation and Later Course.—On December 18 the solid angle was removed down to the labyrinth. No fistula was found. The dura of the middle fossa was elevated, and when the surface of the pyramid beyond the labyrinth was reached pus welled up. On inspection of the pyramid, an irregular opening was found near the posterior edge, and a probe could be inserted to this area.

Convalescence was rapid, and the patient was discharged from the hospital on January 16.

This proved to be a case of pneumococcic infection. The patient undoubtedly had the infection of the pyramid at the time of the involvement of the middle ear in March, and it had existed quiescent from that time until September, when the onset of the labyrinthitis occurred. The involvement of the labyrinth was a retrograde process, developing from the side toward the pyramid and not from the middle ear. The patient made a good recovery but of course will always have a totally deaf ear.

SCLEROMA (RHINOSCLEROMA)

REPORT OF THREE CASES

LEWIS FRANCIS MORRISON, M.D.
SAN FRANCISCO

It would seem that the orthodox procedure in reporting a case or cases of scleroma is to refer to the fact that Hebra [1] first recognized this tumor as a clinical entity and then to refer to the work of von Frisch,[2] who isolated an organism of the Bacillus mucosus capsulatus group, which according to the most modern nomenclature is now known as Klebsiella rhinoscleromatis. There is no reason to recapitulate or paraphrase the works of others. The literature on the subject as regards the still unknown cause, the questionable frequency and the geographic distribution of the lesion is adequate in all respects and is available to any one wishing to have this information. No great steps of progress either in the prevention of the disease or in the treatment of it have been made in several years. Therefore, this paper will be concerned only with the presentation of 3 additional cases of scleroma. The first 2 cases reported present no extraordinary features. The third case is of interest in that the clinical report is supplemented by a record of the autopsic observations and by an excellent drawing. These 3 cases are distinct from those recently reported from this city by Alderson.[3]

REPORT OF CASES

CASE 1.—A woman aged 63, a native of Mexico, was first admitted to the clinic on Aug. 22, 1928, complaining of a pain in the chest. She had entered the United States fourteen years previously. She spoke no English, and even with the help of an interpreter it was impossible to obtain a definite family history. As far as could be ascertained no other members of her family had had any maladies of the nose, the throat or the chest. The patient had definite hypertension and nasal catarrh. A Wassermann test done on the day of her admission to the hospital was negative.

She next returned to the clinic on Nov. 1, 1934, and was seen in the nose and throat department, where examination showed that the nose contained tumor masses which were of sufficient extent almost to obliterate the usual landmarks. The anterior nares were filled with mucopurulent secretion and crusts. The removal of the secretions and crusts revealed irregular smooth-surfaced tume-

From the Department of Otorhinolaryngology, the University of California Medical School.

1. von Hebra, F.: Wien. med. Wchnschr. **20**:1-5, 1870.
2. von Frisch, A.: Wien. med. Wchnschr. **32**:969-972, 1882.
3. Alderson, H. E.: California & West. Med. **47**:317-321 (Nov.) 1937.

factions, which bled freely on receiving the least trauma. In many respects the tissue resembled that seen with tuberculosis of the nose; certain areas were identical in appearance with the "apple jelly" nodules of lupus tuberculosus. Posterior rhinoscopic examination showed huge posterior tips of both inferior turbinated bones. Some islands of tissue which in gross appearance might easily be mistaken for adenoid tissue were observed about both eustachian orifices and on the vault. The pharynx and larynx were normal. Tissue was removed from the area of the middle turbinated bones with a conchotome and was sent for histologic report. In due time the diagnosis of rhinoscleroma was returned. The patient was not seen again, however, until May 7, 1937, at which time she complained again of catarrh and also of a discharge from the right ear. The condition of the nose was not greatly changed. The islands of tissue in the nasopharynx had become a confluent mass. There was a large perforation in the right ear drum, through which protruded a mass of tissue having the same appearance as that present in the nose and nasopharynx. The pharynx and larynx were still normal.

The patient was not seen again until Jan. 22, 1937, at which time the condition of the nose, the throat and the ears was unchanged. She had dyspnea and definite cyanosis. She was sent to the medical department, where physicians noticed a loud systolic murmur and a blood pressure of 220 systolic and 90 diastolic. She was seen in that department at intervals until June 25, at which time her condition was considered improved. The blood pressure was now 195 systolic and 110 diastolic and the pulse rate was 72. A follow-up was attempted but was not successful. A recent report from friends of the patient stated that she is dead and that no postmortem examination was done.

Case 2.—A man aged 28, married, a bus boy and waiter, was first seen in the eye clinic on May 22, 1933, complaining that his eyes watered and that he had a "bad cold." The patient was born in San Salvador, Central America, and lived there until he was 10 years old, at which time he came to California. His wife and two children were living and well.

The family history as obtained from the patient was entirely irrelevant. He had a history of colds and sinus trouble. He had had a bilateral Caldwell-Luc operation at the San Francisco Hospital on Aug. 2, 1929. A note on the operation at that time stated that the right antrum was filled with polypoid mucosa and pus and that the left antrum contained very thick mucous membrane and pus. There was no note as to whether there had been a histologic examination of the tissue; it is logical, therefore, to assume that the tissue had not been sent to a laboratory. Also, one may assume that there was nothing particularly abnormal about the appearance of either the nose or the mucosa of the maxillary sinuses, as there was no note to that effect.

After receiving treatment for the initial complaint, watering of the eyes, the patient was sent to the ear, nose and throat clinic for treatment of the "bad cold." Examination at this time showed that both antrums were filled with what was termed exuberant granulation tissue. This was of sufficient amount practically to occlude both nostrils and had caused the nose to become very broad throughout its entire length. The tissue was not friable, but it bled easily. A biopsy was done. The report, returned June 6, stated in brief that there were atypical epithelial cell hyperplasia and possible carcinoma. In spite of the report the diagnosis did not seem to conform with the clinical picture. The Wassermann reaction was negative. The ears, the nasopharynx and the larynx were entirely normal, as were all other physical findings.

The patient was next seen Dec. 7, 1934, at which time both anterior nares were completely occluded. The nose (externally) was about one and a half times its normal size, and there was a nodular area in the soft tissue of the left cheek, directly over the center of the canine fossa; this looked much like a keloid which might have formed subsequent to the formation of a furuncle. This nodular mass was firmly attached to the bone below. The tissue occluding the anterior nares was firm; it was divided into semisegments by firm bands of fibrous tissue, and it bled profusely on the slightest trauma. Generous amounts of this tissue were removed for histologic examination. It was necessary to pack the area to control the profuse bleeding. The pathologist returned a report of rhinoscleroma. The nasopharynx, the ears and the larynx were normal.

The patient was absent again for a time and was next seen during November 1936, at which time dermatologists were consulted. They suggested a course of autogenous vaccine. The predominant organism obtained was the gram-negative capsulated bacillus. This organism of the Friedlander group was isolated in pure culture, and a quantity of vaccine was prepared. The vaccine was given at biweekly intervals for a period of months, the dose being gradually increased until definite systemic as well as local reactions were obtained. During November some tissue was removed from the nose; it was ground and was injected into guinea pigs. No evidence of either tuberculosis or coccidiosis was observed in these animals.

The only change in the patient during the period of administration of vaccine (other than the systemic reaction and the local reaction about the site of injection) was that the size of the nose steadily continued to increase until by May 1937 it was about twice normal. Shortly after this time the swelling over the bridge of the nose increased rather rapidly and the skin became reddened, and by June 3 it was necessary to open an abscess. About 15 cc. of frank pus was obtained. In two weeks' time all drainage had ceased; the incision closed and healed, and the area subsided so that it was similar to the rest of the surrounding tissues. A second nodular area had appeared slightly below and lateral to the inner angle of the eye. Like its predecessor, it was firmly attached to the bony structure beneath it. The first nodular mass had diminished somewhat, and the skin about it was retracted and firmly bound down to the deeper tissues and to the bone.

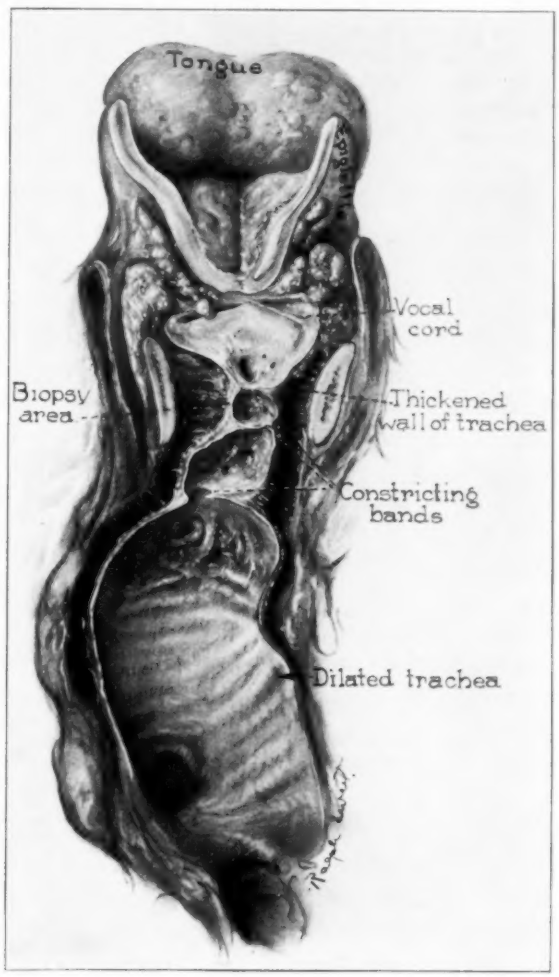
Further consultation with physicians in the department of dermatology resulted in the suggestion that pyrotherapy be tried. There was no objection to this. During the next twelve days the oral temperature of the patient was seven times raised to 41 C. (105.8 F.) and was kept at this point for five hours. The only immediate effect on the patient was a little mental confusion during the artificial fever and for a few hours after it. Secondary results, local or general, had not appeared at the time of writing. Associated with the return of the patient to the ear, nose and throat department was the suggestion that roentgen therapy be tried. Again neither the patient nor I objected. The 200 kilovolt machine was used. Fifteen milliamperes, filtered with 0.5 cm. of copper and 1 cm. of aluminum, was given over a field 10 by 10 cm. at a target distance of 50 cm. The field was masked with lead so that the eyes were protected. Four hundred roentgens were given on June 30, 200 on August 11, 300 on September 3 and 300 on October 1. When the patient was last seen, in December, the size of the nose had diminished by one third, and he was more comfortable than he had been in several years. There was complete cicatricial stenosis of both anterior nares and considerable uplift of the soft palate and uvula due to beginning diaphragm formation at this area.

CASE 3.—A man aged 26, married, was admitted to the clinic complaining of various disorders of the nasopharynx, such as catarrh, dyspnea and hoarseness. The patient was born in Esquintta, Guatemala, Central America, and lived there until he was 21 years old at which time he entered the United States. The family history as obtained from the patient showed that the father had died of dysentery with possible inflammation of the bowels and that the mother was alive and well at the age of 60. One brother and one sister were alive and well; two sisters and one brother were dead. The patient's wife and five children were living and well.

The patient had had malaria beginning at the age of 16 and lasting three years, with no remissions. Crusting and catarrh of the nose had been present for fourteen years; hoarseness had been first noticed two years previously and dyspnea four years previously. There had been inspiratory difficulty; at no time had expiration been difficult. No actual interference with swallowing had been noticed, but the fear of choking caused the patient to swallow carefully and slowly. He had consulted several physicians in Los Angeles, Oakland and San Francisco during the past two years and had also visited clinics in these cities but had received neither relief of his complaint nor information regarding its cause. He had been told that he had ozena and had been given prescriptions for various nasal sprays, ointments and fluids with which to irrigate the nose. He stated that almost everywhere he went blood was taken from his arm and twice fluid was taken from his back and that on all occasions he was told that it was "all right." Subjectively the nasal condition had remained unchanged for several years. The dyspnea on exertion and the difficulty on inspiration were slowly progressive. With the exception of the nose and throat, the physical condition at the time of his admission to the clinic was normal. There was no lymphadenopathy; the blood pressure was 110 systolic and 90 diastolic, and the pulse rate 100. Roentgen examination of the chest showed normal conditions. The Wassermann reaction was negative.

Examination in October 1930 showed both nares to be filled with thick, heavy crusts, which had not nearly so foul an odor as those found in the typical patient with ozena. On removal of these crusts with forceps the mucosa bled slightly from several small areas. This bleeding stopped quickly. Examination of the mucosa showed it to be of uneven contour, containing excoriated areas and many small, flat-topped, slightly raised nodules, which in general appearance were similar to the classic "apple jelly" nodule seen in tuberculous lesions. The mucosa of the anterior aspects of the inferior and middle turbinated bones was hypertrophic and revealed much less visible evidence of pathologic change. Both posterior choanae were diminished in size and showed definite diaphragm formation. The septum was thick and high; there was a small spur low to the right, but in general the septum occupied a middle position; it was not a cause of impaired drainage or ventilation. The condition of the teeth was only fair. The tonsils were not involved. There was an almost complete diaphragm from the upper posterior portions of the posterior pillars and the soft palate to the lateral and posterior pharyngeal walls, which separated the epipharynx or nasopharynx from the true pharynx except for an opening approximately 1.5 cm. in diameter slightly to the right of the midline. The hypopharynx, or epiglottis, was normal in color and appearance. The arytenoid cartilages, the true cords and the false cords were normal. There was no evidence of induration, ulceration or infiltration. There was no true paresis or paralysis, but there was a definite limitation of motion on deep inspiration and on attempted phonation. Approximately 1 cm. below the vocal cords was a diaphragm which had a central oval

opening. This opening measured about 1 by 0.5 cm., the long diameter being in the anteroposterior plane. There was a second diaphragm about 1.5 cm. below. On direct examination with a 5 mm. bronchoscope a third diaphragm was seen about 1 cm. below. Since its opening was relatively small and the other openings were eccentrically placed, it was deemed prudent to forego employing the force which would have been necessary to aline these openings because the



Autopsy specimen from patient with rhinoscleroma.

lumen of the trachea between the first and second and second and third diaphragms was less than half normal and the walls were rigid (see drawing).

This patient was seen from time to time. His appearance at the clinic was usually of his own choice and occurred during or immediately after an acute cold. These acute infections of the upper part of the respiratory tract were always accompanied by more than the usual tracheobronchial involvement, as

is common in persons in whom the mucosa of the upper part of the respiratory tract has been in poor condition for many years. The effort of coughing was very tiring, and worse still was the fear of choking to death from temporary obstruction of one or the other of the constrictions by crusts forming over the lumen. On Nov. 5, 1936, roentgen therapy was suggested. One treatment was given, but further therapy was refused by the patient.

On July 30 the patient entered the city hospital gasping for breath. After a short examination the resident physicians decided that a tracheotomy was essential. This was done in the customary area, and a no. 5 tube was inserted after considerable difficulty in gaining entrance to a lumen through the thickened tracheal wall. The respirations were less difficult, but the patient died the same day because of cardiac rather than respiratory failure. Fortunately his stay in the hospital was of such short duration that a postmortem examination was demanded and done by the coroner's office. The specimen shown in the drawing was obtained. There was no evidence of the disease except the lesions in the respiratory tract.

COMMENT

Chamberlin [4] made the statement that there are three stages of the disease, the first, or catarrhal, stage, characterized by increased secretion and formation of crusts; the second stage, that of tumefaction characterized by proliferation, and the third stage, that of cicatrization and concentric stenosis. From the purely clinical standpoint these divisions are not quite so clearcut; even these few cases show that there is considerable overlapping of the stages. Histologic examination of the various specimens showed overlapping of the second and third stages.

Case 2 is of interest in that various measures were employed with no benefit to the patient. The results obtained with roentgen therapy are encouraging. Canfield [5] believed that the benefit obtained with this type of therapy rests in the fact that the roentgen ray matures the lesion and masters the third stage. This is probably the correct analysis and deduction. It is planned to give this patient additional roentgen treatment, approximately 300 roentgens at intervals of four weeks, until there is no further advantage in continuing the procedure.

Case 3 has several points of interest. First, the patient had spent more than two years of time and all his available surplus funds in an endeavor to ascertain the nature of his malady and had not been enlightened. Second, during his pilgrimage he had been seen by a few first class physicians and several well trained ones, who failed to recognize the condition. Were it not pathetic, the plight of the patient during this time would almost be humorous, in that in each instance he usually went through the following routine: Treatment for catarrh for two or three weeks, supplemented with inhalations at home of steam plus certain of the aromatic oils; then roentgen examination of the accessory

4. Chamberlin, W. B.: Rhinoscleroma: Is It an Indigenous Disease? Arch. Otolaryng. **23**:285-294 (March) 1936.

5. Canfield, N.: Ann. Otol., Rhin. & Laryng. **42**:903-908 (Sept.) 1933.

nasal sinuses, with subsequent irrigations, and finally a Wassermann test, with its uniformly negative result. Then the patient ran out of funds or confidence or both and rested a short period to replenish either or both. The third point of interest is the larynx and the tracheobronchial tree as seen at postmortem examination. The apparent absence of pathologic processes in the larynx proper is most striking when one remembers the advanced state of the nasopharyngeal lesion and then sees the thickening of the walls of the trachea and the dense cicatricial diaphragms encroaching on its lumen. The dilatation of the lower one third of the trachea and the right and left main bronchi shows plainly that the ingress and the egress of air were obtained only by great and sustained effort.

Histologic examination of tissue removed from the area marked in the drawing showed extremely dense fibrous or cicatricial tissue, in which were sparsely distributed small islands of typical scleromatous tissue. The islands of scleromatous tissue were so small and relatively so infrequent that it would have been easy to miss them entirely or to fail to recognize them as being of any importance unless the clinical diagnosis had been sent with the specimen. The tumefied tissue seen in the second stage produces slides that are most characteristic of the disease. Haphazard fields selected at any part of the sections produce slides similar in all respects to the textbook pictures. Therefore, it is important that a little thought and a moderate amount of care be exercised in selecting suitable material for biopsy.

SUMMARY AND CONCLUSIONS

Three cases of scleroma are presented, 1 with autopsic observations.

In 1 patient, treated energetically by three methods, roentgen therapy gave the best results.

It is evident that the diagnosis of scleroma depends on adequate and properly selected material for biopsy.

It is important to remember the possibility of the presence of scleroma in patients who have chronic lesions of the nose, the pharynx, the larynx or the trachea that in many respects simulate those of ozena, syphilis, the granulomas or the malignant tumors, individually or combined.

Diaphragm formation and concentric stenosis are characteristic only of the third stage of this disease.

COMPLICATIONS FOLLOWING RHINOPLASTY

ARTHUR PALMER, M.D.
NEW YORK

It is natural for the surgeon to exhibit his cured patient with a feeling of pride. He has succeeded in the face of obstacles, and his labors have been rewarded. He presents his case before a medical society with photographs, slides or motion pictures and a recitation of how the operation was performed and receives the congratulation of friends and fellow members. All of this is stimulating and perhaps food for vanity. But what of the failures and the complications? Are they passed off lightly or ignored completely? May the surgeon take to himself the credit for success and pass on to the processes of nature, to the hospital, to the nursing staff or to the anesthetist the blame for failure?

The wise surgeon profits more by his failures than by his cures. He examines the causes of failure with diligence and with diligence seeks their correction. Complications may arise from a rhinoplastic operation just as they may from any other type of operation, and it is worth while indeed to classify these complications, study their causes and discuss the means of avoiding them.

It appears from the examination of books and articles on plastic surgery that the complications associated with surgical procedures of this type have received slight attention—far less than their incidence or their importance warrants. A review of the literature on plastic surgery reveals only occasional paragraphs dealing with complications, and these are concerned largely with complications following the injection of paraffin, a method which is obsolete.

Rhinoplastic complications ranging from minor infections to death may be classified as follows:

1. Infections, local or distant
    - Cellulitis
    - Abscess (with loss of transplant)
    - Sinusitis
    - Pharyngitis
    - Tonsillitis
    - Pyelitis
    - Arthritis
    - Flare-up of chronic tuberculosis
    - Parotitis
    - Thrombophlebitis
    - Septicemia

Read at the Annual Meeting of the Society of Plastic and Reconstructive Surgery, St. Louis, Oct. 22, 1937.

Pulmonary complications of infectious origin
- Bronchitis
- Bronchopneumonia
- Infarction
- Embolism
- Pleurisy
- Pulmonary abscess
- Empyema
- Atelectasis

2. Structural or mechanical complications
   - Narrowing of the nares
   - Loss of the sense of smell
   - Difficult breathing
   - Interference with the normal respiratory functions of the nose by damage to the mucous membrane
   - Cosmetic defects due to faulty technic, such as cutting through cartilage of the columella

3. Circulatory complications
   - Hemorrhage
   - Shock
   - Induration of wounds (devitalized)
   - Hematoma
   - Gangrene
   - Localized thrombosis (in region of the wound)
   - Embolism
     - Fat
     - Gas
   - Infarction
   - Metabolic (chemical)
     - Acidosis
     - Hyperglycemia
     - Absorption of transplant, either bony or cartilaginous

4. Psychic complications
   - Psychoses ranging from simple depression to mania

Postoperative complications other than those mentioned, such as paralytic ileus, abscess of the liver, dilatation of the stomach, retention of urine and nerve paralysis, occur more commonly after surgical procedures on other parts of the body than after operations on the nose. I am not concerned with immediate effects of the anesthetic, such as cocaine poisoning, or with respiratory failure resulting from general anesthetic.

INFECTIONS

In cases of postoperative infection the burden of responsibility rests on the nurses in the operating room, on the surgeon and on his assistants. Blaming the suture and ligature materials is rarely justified. Many

infections arise from a preexisting infection in the patient which has been activated by the ordeal and trauma of the operation. Fortunately, the skin of the face is resistant to infection, and rhinoplastic infections are comparatively rare. It is a mistake to think of any operative wound as sterile, that is, as completely free from micro-organisms. In all of them the resistance of the tissues is pitted against a certain amount of contamination; if the contamination exceeds this degree of resistance, infection results (Harrison [1]).

With reference to submucous resection of the septum, Laszlo [2] pointed out that the removal of cotton or gauze swabs and of small fragments of bone and cartilage is important in preventing hematoma and secondary infections. Suppuration in the middle ear may occur after nasal operations, owing to the packing or to forced blowing of the nose. Similarly, an infection of the throat such as tonsillitis and acute pharyngitis due to mouth breathing or possible stagnation of mucus in the postnasal spaces sometimes complicates nasal operation.

Most surgeons agree that if infections occur and lead to formation of an abscess, drainage may be obtained from within the nose by reopening the incision leading to the site of the pocket and inserting a drain. Should the abscess form at a considerable distance from the point of incision, it is necessary to drain it externally by making an incision over its site. Nonlocalizing infections are treated in the usual manner, as if no intranasal operation had been performed (Safian [3]).

REPORT OF CASES

CASE 1.—A man aged 41 suffered from a "nervous breakdown" in 1926, at the beginning of which the question of the presence of incipient pulmonary tuberculosis was raised.

Examination showed the presence of an external nasal deformity characterized by a twisted dorsum, large and asymmetric nostrils, deviation of the nasal septum to the right and dislocation of the anterior portion of the columella. The turbinated bones were generally hypertrophic. The deformities of the nose were of traumatic origin.

Operation was performed on March 26, 1937, with the region under local anesthesia. It consisted of: (1) submucous resection of the septum; (2) clipping of the inferior turbinates and removal of their posterior poles; (3) reduction of the middle turbinates by splitting them and removing the lateral half; (4) rhinoplasty, and (5) mobilization of the fractured bones, rendering symmetric the nostrils and the nasal tip by reducing the right alar cartilage. A piece of cartilage was implanted just above the right alar cartilage to fill out a depression in the right side of the nose.

---

1. Harrison, P. W.: Postoperative Infections, Ann. Surg. **104**:316 (Aug.) 1936.

2. Laszlo, A. F.: Practical Points on Submucous Septum Resection: Pitfalls and Corrections, Laryngoscope **46**:840 (Nov.) 1936.

3. Safian, J.: Corrective Rhinoplastic Surgery, New York, Paul B. Hoeber, Inc., 1935, p. 7.

During the intranasal operation excessive bleeding was encountered, in spite of local anesthesia. Tight intranasal packing was used, and an external fixing bandage was applied.

The postoperative local reaction (ecchymosis and swelling of the face) was marked, but it began to subside on the fourth day.

The packing was left in the nose for four days. Nevertheless, its removal was followed by excessive bleeding, which lasted about twenty minutes. On the fifth day the patient was removed to his home for convalescence.

At this time hemorrhages of moderate severity occurred, which finally made repacking necessary. Ten days after the operation the patient, although weak, resumed his professional work. On the thirteenth day he woke in the morning with chills. His temperature rose to 101 F. He had a sore throat and felt slight itching and moderate soreness on the tip of the nose, where, also, a small red spot appeared. In twenty-four hours the temperature was almost normal, but slight soreness and some swelling at the tip of the nose persisted. Boric acid dressing was applied to the nose. A slight oozing of blood began at this time and remained constant. Two and one-half days later the swelling of the nose began to increase rapidly, and within a few hours it spread over both eyelids and the central part of the forehead, and intranasally as well. Headaches, general malaise and considerable secondary anemia developed.

The presence of pus was suspected, but extensive probing, with incisions made both externally and intranasally, did not yield pus. The patient's general condition gradually became worse, and on April 13 he was readmitted to the hospital.

At this point I found the patient acutely ill. The face was swollen, the nose being enlarged to one-half the size of a man's fist. There was moderate bleeding from the external and intranasal incisions.

Examination of the blood showed: hemoglobin content 64 per cent, red cells 3,800,000, white cells 20,000, adult polymorphonuclear leukocytes 85 per cent and lymphocytes 15 per cent. The bleeding time was one and one-half minutes, and the coagulation time, eight minutes. The Wassermann and Kline reactions of the blood were negative. The blood culture was sterile after five days. The temperature was 38 C. (100.4 F.), and the pulse rate, 108.

The following treatment was instituted: Dressings soaked in a saturated solution of magnesium sulfate were applied locally. Injections of 5 cc. of prontosil (the disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonic acid) were given intramuscularly three times a day and 0.3 Gm. of sulfanilamide by mouth twice a day. Laxatives were given as needed, and a high vitamin diet was ordered. Pentobarbital sodium and opiates were used. Drops of a 10 per cent solution of mild protein silver were instilled in the eyes.

On April 14 a blood transfusion (250 cc.) was given. The eyes were irrigated with boric acid and a 1 per cent solution of ephedrine in oil was given intranasally. The urine at this time turned red and hazy.

On April 16 the blood picture showed improvement. The count was: hemoglobin content 80 per cent, red blood cells 4,500,000, white blood cells 7,000, adult polymorphonuclear leukocytes 57 per cent and immature polymorphonuclear leukocytes 40 per cent.

From this point the patient's condition showed steady improvement. The swelling of the nose and eyes was practically gone. On April 18 a seropurulent discharge was present in the right eye. This subsided gradually when irrigations of boric acid and drops of mild protein silver were used. For the residual nasal and facial swelling warm compresses of boric acid were applied. The temperature

gradually returned to normal. On April 23, after steady improvement, the patient was discharged. He was advised to have further rest, as he was still weak.

The patient regained complete health after two weeks of further convalescence and is in good health at present. The result of the nasal operation is satisfactory.

A case illustrating a pulmonary complication was particularly interesting.

CASE 2.—After a transplant of costal cartilage for saddle back deformity of the nose, the patient's temperature rose to 103 F., with development of signs of involvement of the bases of both lungs posteriorly. Dulness was present, as were moist and sibilant rales. This condition cleared up promptly after removal of the tight band of adhesive plaster which encircled the chest as part of the dressing. It is probable that there had been a collection of infected mucus in the lower parts of both lungs, due to interference with the normal expansion of the wall of the chest and the inability of the patient to expel this secretion by coughing. The adhesive plaster was strapped so tightly that it interfered with the normal physiologic functions of the thoracic wall.

After this experience, it has been my custom to apply a simple dressing over the thoracic wound, which causes no difficulty in respiration.

Gillies [4] reported a case in which the patient had three operations, for the correction of a wound caused by an injury from a Verey light. The tagliacotian method was used.

On the third day following this [the third] operation a severe attack of erysipelas occurred, followed by a small amount of local gangrene and septic bronchopneumonia, from which the patient died in an isolation hospital.

STRUCTURAL OR MECHANICAL COMPLICATIONS

Before rhinoplastic operation is undertaken, one does well to consider the possibility of structural and mechanical complications. A thorough understanding of the anatomic and physiologic relations of the nose is essential. Many times the surgeon, in his eagerness to improve the form of the nose, has impaired its function. Narrowing the nasal bony wall may cause partial obstruction and interference with respiration. Destruction or trauma of the mucous membrane may result in loss of the sense of smell, and deformities of the nasal tip and the columella will surely follow ill advised section or resection of the alar cartilage and the cartilage of the columella.

Laszlo [2] pointed out that anosmia may be temporary or permanent. Temporary anosmia usually disappears after a few days, when the swelling has gone from the nose. In cases of permanent anosmia the olfactory region has received some injury. Meningitis, one of the dreaded compli-

4. Gillies, H. D.: Plastic Surgery of the Face Based on Selected Cases of War Injuries of the Face, Including Burns, New York, Oxford University Press, 1920, p. 254.

cations of submucous resection of the septum, is usually of short duration. The causes are not definitely known, but the condition is usually due to a severe infection or possibly to an injury to the cribriform plate.

One of the most common complications following rhinoplasty is perforation of the septum, due to rough or unskilled work. If the perforation is small plastic repair can be done, but if it is large correction will be almost impossible. Laszlo [2] stated further that a late complication of submucous resection of the septum is dropping of the bridge of the nose (saddle nose), due to faulty removal of the cartilage and bone from between the nasal bones. This dropping will not be observed for months and sometimes not for years after the operation. Plastic repair is then indicated.[2]

CASE 3.—F. K., a woman aged 23, had a traumatic deformity of the nose. She had fallen at fire drill in school, at the age of 10 years, and had sustained a fracture of the nose which resulted in lateral displacement to the right, a dorsal hump, prominence of the columella and high deviation of the septal cartilage to the left. A nasal plastic operation was performed on Nov. 13, 1936. The nasal bones were mobilized after removal of the dorsal hump. The deformity in the region of the columella was corrected. After the operation the left lateral bony wall of the nose was so close to the septum that interference with breathing occurred on the left side.

CIRCULATORY COMPLICATIONS

The most common of the circulatory complications in rhinoplasty is hemorrhage. If no epinephrine is used this may be very troublesome to the surgeon during the operation, though it is seldom serious to the patient. A variety of instruments for nasal and throat suction should be at hand. Packing is seldom used during the operation. A properly applied splint will go far to prevent postoperative hemorrhages under the skin, and at times intranasal packing is indicated. This packing should be removed in twenty-four hours. Secondary hemorrhage occurring several days after an operation is usually associated with infection, the presence of which complicates the treatment.

Bleeding while operating is one of the most common complications that prevent satisfactory work and may be caused by: (1) A little or no adrenalin used; (2) separating the wrong cleavage; (3) tearing of the mucous membrane (page 843).[2]

Laszlo also remarked:

Hematoma formations may take place in the first 24 hours in cases where no packing is applied, and later in cases where packing was applied over night. A small clot or piece of cartilage or bone between flaps may cause these hematomata. . . . To prevent this, an opening in the flap of the floor of the nose will help. If a hematoma has formed, an opening should be made either in the original incision or on the floor of the nose; this will usually remedy the situation

although the hematoma is apt to recur. If large hematomata are present a secondary packing (Simpson, Burney splints) may be necessary. If the hematoma is not opened it may break down and cause septal abscesses, or remain partly absorbed, causing permanent thickening of the septum and, therefore, further complaints. Too much clearing of the nose or suction after the removal of the first packing is not a practical procedure, for the healing will be retarded and may cause late infection or other complications.

In contrast to hemorrhage, shock is rarely seen complicating plastic operations on the nose. Lowering of the blood pressure is the characteristic feature of shock, with diminution of the volume of circulating blood. Hemorrhage or drug poisoning may be the cause. In the early recognition of hemorrhage and shock I agree with Graham [5] that "nothing can take the place of the frequent determination of the patient's condition by the surgeon himself."

The length of this paper will permit no more than mention of the less common complications such as thrombosis.

PSYCHIC COMPLICATIONS

That mental complications follow rhinoplastic operations is not surprising. The sensitive patient often has an inferiority complex and lacks confidence, having been subjected to teasing and ridicule by thoughtless acquaintances. Again, friends and relatives, when consulted, have advised against the operation, so that the patient may have had a conflict of emotions for many months before he finally decided to consult a surgeon. Reactions following the operation may exhibit themselves in all degrees of psychosis, even to a state of mania.

Graham [6] said:

It is quite safe to state that fear psychoses and their sequelae do not occur in individuals with normal nervous mechanism. The importance of recognizing the condition before operation is well known to experienced operators.

It is often advisable to have the patient consult a psychiatrist when there is doubt that his mental reaction following the operation will be normal. The advice of a psychiatrist in regard to operation should be followed if one wishes to avoid unpleasant psychic complications.

CASE 4.—R. W., a woman aged 23, came to the clinic to have a nasal hump removed surgically. Her nose had been broken at the age of 6 years. Her only complaint was that her nose usually bled before her menstrual periods, the flow lasting about two minutes. The skin across the nose was tight and painful and was apt to crack in cold weather because of the broken bone.

5. Graham, E. A.: Surgical Diagnosis, Philadelphia, W. B. Saunders Company, 1930, p. 139.

6. Graham,[5] p. 159.

An operation was performed on Nov. 13, 1934, with the use of local anesthesia. Intranasal incisions were made; the dorsal hump was removed with saw and rasp; the lateral nasal bony wall was mobilized by sawing through the nasal processes of the superior maxillas, and the nose was shortened by removal of part of the septal cartilage at its base. The right alar cartilage was resected by a cutaneous incision at the lower border on the right side.

The patient was pleased with the result.

Several weeks after the operation mild depression, commonly described as a "nervous breakdown," developed, which necessitated rest and absence from home for seven weeks. The patient made a complete recovery.

Consideration of the complications of rhinologic surgical procedures should stimulate interest in prophylaxis. During recent years there has arisen the tendency to study end results. The follow-up clinic in the surgical department of many hospitals is well organized. Here it has been learned what grave sequelae may follow simple operations. As a result, the patient has been better prepared for the operation, and the lapses in surgical technic have been fewer. It is known that starvation and dehydration by severe purgatives cause acidosis. High fluid intake is essential in postoperative care. The physician has learned to disturb the patient as little as possible in the preoperative regime.

Cutler and Scott [7] pointed out that the favorable postoperative course of patients submitted for emergency operation with little or no preparation amply proves the wisdom of the method just described, that is, as little interference as possible with daily routine prior to operation, even to the extent of concealing the exact time of the operation in the case of an apprehensive, high-strung patient. Volkmann emphasized the importance of a careful study of the nervous system and the institution of proper environmental conditions. The study is well repaid in the lessening of postoperative mental complications.

CONCLUSION

Complications following rhinoplastic procedures are not common, but on occasion a great variety of complications may occur. Care in preoperative preparation of the patient as regards both physiologic and mental factors is important. Strict surgical technic will prevent other complications from occurring, and constant attention to the patient after the operation is of great aid in making an early diagnosis when complications occur.

7. Cutler and Scott, cited by Graham.[5]

OTOGENIC ASPECTS OF ARACHNOIDITIS

S. L. SHAPIRO, M.D.
CHICAGO

Among the more unusual neurologic conditions which may concern the otologist are certain abnormal accumulations of cerebrospinal fluid variously termed "meningitis serosa circumscripta,"[1] "meningeal hydrops,"[2] "otitic hydrocephalus"[3] and "arachnoiditis."[4] The cases recorded in the literature, although showing considerable variation as to the origin, course and symptoms of the condition, nevertheless have certain points in common. The clinical picture as a rule suggests some serious intracranial condition or complication, demanding surgical treatment. There is uniformly evidence of increased pressure; the cerebrospinal fluid either is normal or shows only a mild meningitic reaction, while the simple relief of pressure either by operation or through other means generally effects a rapid and permanent cure. While the pathogenesis is by no means clear in every case, the majority of those observations which were made at operation or after histologic study indicated changes in the arachnoid membrane. The term arachnoiditis will therefore be used as a general name for the conditions described.

ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

The Arachnoid Membrane.—It will be recalled that the meninges of the brain consist of three layers: the dura mater, or outermost covering, which is closely adherent to the inner surface of the cranial bones and is also reflected to form certain rigid septums which project into the cranial cavity; the arachnoid membrane, separated from the dura mater by a potential cavity known as the subdural space, and the innermost layer or pia mater, which is closely applied to the surface of the brain and follows its every fold and convolution. The arachnoid is a delicate

---

Read before the Chicago Laryngological and Otological Society, March 7, 1938.

From the Department of Otolaryngology, University of Illinois College of Medicine.

1. Cushing, H.: Tumors of the Nervus Acusticus, Philadelphia, W. B. Saunders Company, 1917.

2. Passot, R.: Meningites et états meninges aseptiques d'origine otique, Paris, G. Steinheil, 1913.

3. Symonds, C. P.: Otitic Hydrocephalus, Brain **54**:55, 1931.

4. Horrax, G.: Generalized Cisternal Arachnoiditis Simulating Cerebellar Tumor: Its Surgical Treatment and End-Results, Arch. Surg. **9**:95 (July) 1924.

membrane of connective tissue, which is connected by numerous trabeculae to the underlying pia mater, the space between these two meningeal layers being known as the subarachnoid space. Both the inner aspect of the arachnoid and the trabeculae are lined with flattened mesothelial cells.[5] The blood vessels supplying the brain and spinal cord traverse the stroma of connective tissue of the pia-arachnoid, forming rich networks of small arteries and veins. Since the arachnoid, unlike the pia mater, does not follow closely the contours of the brain but rather the inner aspect of the dura mater, the subarachnoid space is much wider in some places than in others. The dilated areas are known as cisterns, the most important ones being at the base of the skull. They are arranged in a somewhat complicated manner, with numerous extensions and communications with each other and with the rest of the subarachnoid space.

The Cerebrospinal Fluid.—Probably formed by a process of filtration from the blood, chiefly through the choroid plexuses in the lateral ventricles, the cerebrospinal fluid passes out from each ventricle by means of the interventricular foramens into the third ventricle and from there into the fourth ventricle by way of the aqueduct of Sylvius. Its further passage is through the two lateral foramens of Luschka and the median foramen of Magendie, in the roof of the fourth ventricle, into the cisterna magna. Thence the fluid spreads around the brain stem, cerebral hemispheres and spinal cord. Important cisterns at the base besides the cisterna magna are the cisterna pontis, on the ventral aspect of the pons, and especially its lateral prolongations, which occupy the cerebellopontile angles and are adjacent to the petrous pyramid; the cisterna interpeduncularis, which occupies the space between the cerebral peduncles anterior to the pons, and the chiasmic cistern, in the region of the optic chiasm.

A definite circulatory cycle of the cerebrospinal fluid is known to exist throughout the subarachnoid space. Absorption of the fluid takes place mainly through the arachnoid villi, which project through the walls of the venous sinuses into the blood stream. It must be stated that to some extent both formation and absorption of cerebrospinal fluid may take place in other ways than the ones just described,[6] particularly under abnormal conditions.

The Temporal Bones.—With the exception of the occipital bone, the greatest amount of surface contact with the dura of any bone at the

5. Kuntz, A.: Neuro-Anatomy, Philadelphia, Lea & Febiger, 1936.

6. Plaut, F.: Normale und pathologische Physiologie des Liquor cerebrospinalis, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und patholgischen Physiologie, Berlin, Julius Springer, 1927, vol. 10, pp. 1195-1199.

base of the skull, as well as the largest number of possible pathways for creating intracranial complications is presented by the temporal bone. Anteriorly and superiorly, the roof of the attic, the antrum, the mastoid cavity and the petrous apex offer a threat to the cerebral meninges; posteriorly, the sinus, the mastoid cells, the labyrinth, the internal auditory meatus, the fossa subarcuata and the saccus endolymphaticus are a source of danger to the dura of the cerebellum and the cisterns of the posterior fossa.

CAUSATION AND PATHOLOGIC PICTURE

Frequency.—It is impossible to state the frequency of arachnoiditis with any accuracy. Horrax [4] in 1924 reported 33 cases studied in a ten year period in Cushing's clinic, during which time 676 cases of verified tumor were observed. In a recent monograph on the subject, a group of French writers [7] were able to give details of 66 cases. From the number of scattered reports and comments on the condition by neurosurgeons, neurologists and otologists, one gets the impression that arachnoiditis is found more commonly than is generally supposed.

Causes.—Arachnoidal inflammation may result from trauma to the head, from disease of a contiguous bone, from a generalized infection, such as scarlet fever or influenza, or from a disease of the central nervous system, such as cerebrospinal meningitis, encephalitis, syphilis or multiple sclerosis. In the middle and especially in the posterior fossa all other causes are of subordinate importance to infection of the ear. This may be acute or chronic; even catarrhal otitis media has been considered as an etiologic factor.[7] In many recorded cases the possible focus in the ear had long since cleared up by the time the symptoms of arachnoiditis became manifest.

Of the causative organisms in the adjacent focus, the streptococcus and the pneumococcus of type III are said to be the most common. As the fluid is nearly always sterile, it is assumed that only the toxins reach the arachnoid membrane. "Minimal virulence," just enough to produce a mild inflammation, is therefore requisite for the occurrence of arachnoiditis.

Classification.—From a pathologic standpoint the following forms occur:

1. A fibrous and adhesive variety. This is common in the anterior but uncommon in the posterior fossa.

2. A cystic form. In this some part of the subarachnoid space, usually one of the cisterns, undergoes mild inflammation, resulting in

7. Bourgeois, R., and others: Les arachnoïdites de la base du cerveau, Rev. d'oto-neuro-opht. **14**:321, 1936.

obstruction of the normal circulation of the cerebrospinal fluid, with formation of a pseudocyst in the affected area. Grossly the arachnoid membrane is thickened and opaque; histologically there is infiltration of round cells in the arachnoid mesh and in some cases sclerotic changes in the underlying cortex.[8]

3. Internal hydrocephalus. This may be due to (*a*) inflammatory closure of one or more of the foramens in the ventricles, the aqueduct of Sylvius or the roof of the fourth ventricle, or (*b*) hypersecretion with so-called mechanical obstruction of the foramens.

4. Generalized intracranial hypertension. There is a free flow through the subarachnoid space, but the condition is due to (*a*) hypersecretion or (*b*) obstruction to the normal absorptive channels by inflammation of the arachnoid villi.

SYMPTOMS AND COURSE

From a clinical aspect cases of arachnoiditis fall into three groups:

A. Cases in which the history and symptoms suggest a tumor of the brain.

B. Cases in which abscess of the brain appears probable.

C. Cases in which there is a relatively sudden rise of intracranial pressure with severe symptoms but with no focal signs.

All three groups may possess an interest for the otologist, both from an etiologic and from a diagnostic point of view.

Pseudotumor of the Posterior Fossa Due to Arachnoiditis.—As an example of the first type of arachnoiditis the following case is reported:

A white girl, single, aged 18, was admitted to the dispensary on Oct. 8, 1937, with complaints of pain in the chest and back of one month's duration and severe headache, dizziness, nausea and vomiting for the past three months. Examination disclosed no definite findings beyond probable pregnancy. Because of some abnormalities in behavior she was referred to the psychiatric department, where a diagnosis of probable early paranoid schizophrenia was made; later some nystagmus was noted, and because of this as well as the complaint of tinnitus she was referred to the ear, nose and throat department, where she was examined on October 27 and again on November 4.

A history of a previous discharge from the left ear in childhood was elicited; the patient complained of tinnitus in both ears but more in the left. The tympanic membranes were retracted but intact. The nose and throat were normal. Functional tests indicated that the hearing of the right ear was essentially normal both for the whispered voice and for the tuning fork. In the left ear the whispered voice was heard at 10 feet (3.3 meters); the reaction to the Schwabach test was normal; function, according to the Weber test, was not lateralized; the Rinne test elicited a short positive reaction; the hearing for low tones was somewhat dimin-

8. Davis, L., and Haven, H. A.: A Clinicopathologic Study of the Intracranial Arachnoid Membrane, J. Nerv. & Ment. Dis. **73**:129 and 286, 1931.

ished, and the hearing for high tones was about half of that in the other ear. Vestibular tests were made, with the following results: there was sustained spontaneous nystagmus when the patient looked to either side but no 2 degree nystagmus even under the Frenzel glasses; there was a slight spontaneous past pointing to the right, but neither Romberg's sign nor deviation in gait with the eyes closed was observed. The caloric test elicited a normal response as to nystagmus and past pointing on the right side; no vestibular response whatever was observed on the left side.

A diagnosis of a lesion of the posterior fossa was made and the patient was referred to the department of neurologic surgery, where additional findings of a choked disk of from 1 to 1½ diopters, absent corneal reflex on the left side, slight slowness and awkwardness of the left hand, slight ataxia and slight increase of the deep reflexes of the left arm and leg were noted. The diagnosis by the department of neurosurgery was tumor involving the left cerebellopontile angle, most likely an acoustic neuroma. A history of a fall on the head, with some unconsciousness, six years before was also obtained. Roentgenograms of the skull showed no pathologic condition.

An operation, performed on December 2 by Dr. Oldberg, showed cystic arachnoiditis with no other abnormality or tumor in the left cerebellopontile angle. The patient made an uneventful recovery and has been seen since. She is in an advanced state of pregnancy but feels well otherwise, although giving the impression that she is rather psychotic. She still has some tinnitus in the left ear, and the caloric reaction continues to be absent, but the hearing, particularly of the tuning forks, has markedly improved. There is still some spontaneous nystagmus as she looks to each side.

The diagnosis in this case was made more difficult by the pregnancy and the psychotic personality of the patient. The latter finding has been noted by other observers in patients in this condition. From the otologic standpoint the diagnosis of a lesion of the posterior fossa was made on the basis of dissociation of the eighth nerve of a central type as well as of spontaneous nystagmus of a central type. Although the condition suggested an acoustic neuroma, the preservation of a relatively good hearing without vestibular reaction was so unusual with this condition that it was felt no specific diagnosis could be made.

The great majority of the reports of pseudotumor of the posterior fossa due to arachnoiditis have been submitted by neurosurgeons and neurologists. Cystic dilatation of the lateral pontile cistern simulating tumor of the cerebellopontile angle is the most common condition of this type, probably because this cistern is the one most directly exposed to infection from the temporal bone. The symptoms are generally similar to those in the case just cited. A slow onset is the rule, often after some mild ailment of the ear, which has since been forgotten, with headache, dizziness, tinnitus and hypoacusis. Neuralgia, facial weakness or spasm or paresis of the sixth nerve may occur; cerebellar signs are inconstant. Vestibular findings may be unilateral loss of function combined with loss of hearing or a dissociation of the type noted in my case; Bourgeois and his associates [7] stressed the latter as a sign pointing

to arachnoiditis. Mullin [9] found no responses from the vertical canal on either side in 1 case. Nearly all observers comment on the difficulty of differentiating the condition from a tumor—a difficulty which is increased by the fact that a tumor in this location is often accompanied by a dilated cistern.

More rarely cystic arachnoiditis in the posterior fossa may be localized in the internal auditory meatus, the anterior aspect of the cerebellum, the cerebellar hemisphere or the ventral surface of the pons. Focal symptoms suggestive of these locations are characteristically less pronounced than the signs of hypertension.

Cases in Which Abscess of the Brain Is Simulated.—As an example of the second type of arachnoiditis the following case is reported:

A white child, aged 6 years, was seen at the pediatric service on Jan. 6, 1936, with the complaint of headaches for the past two weeks and intermittent vomiting for one month. There was a history of an intermittently discharging right ear for the past three years; the present illness set in about a month previously with a high fever, the symptoms just noted, loss of appetite and loss of weight. The patient when admitted was drowsy; there was hyperesthesia; Kernig's sign was present, and Brudzinski's was suggested. Spinal puncture showed the fluid under increased pressure; it was cloudy, gave a positive reaction to Pandy's test and had a leukocyte count of 1,500, with from 90 to 94 per cent neutrophils. The leukocyte count of the blood was 8,000, with 67 per cent neutrophils. The diagnosis of the ear department was exacerbation of chronic otitis media with possible intracranial involvement. At operation, on January 8, the mastoid was found to be sclerotic but bled more than normally. Two large cells in the tip were full of a gelatinous material. The sinus and dura of the posterior fossa were exposed for a large area and found to be normal. The dura of the middle fossa was exposed for about 2 cm. and found to be considerably injected. A radical mastoid operation was completed and the wound left partly open. Cultures of secretion taken from the middle ear before operation revealed Staphylococcus aureus and Streptococcus viridans; cultures of material taken from the dura of the middle fossa at operation were negative; cultures of the spinal fluid showed an organism resembling a meningococcus, which, however, could not be obtained at subsequent spinal punctures.

After the operation the child did not do well. Both the drowsiness and the vomiting continued, as well as low grade fever; the spinal fluid, however, showed fewer cells. Six days after the operation I felt that the presence of an abscess of the brain or encephalitis was still to be determined. The patient continued to be treated by the pediatric department, with the symptoms as stated and increasing loss of weight, although at times there were slight remissions. On February 5 a pure horizontal nystagmus to the right, of irregular amplitude and of a sustained character, was noted; but this time mild choking of the disk had also developed. The patient was seen by me on this date and on February 11, when a diagnosis of abscess of the temporal lobe was made and exploration advised. The neurosurgical department decided that a cerebellar abscess was present and advised further waiting for localization. Exploration by Dr. Oldberg on February 29

9. Mullin, W. V.: Circumscribed Arachnoid Cyst Giving Symptoms of Acoustic Neuroma, Laryngoscope **43**:213, 1933.

revealed no abscess in the cerebellum. On March 4 the temporal lobe was explored by Dr. Oldberg through the squama. A dilated right ventricle was found but no pus. A diagnosis of internal hydrocephalus on the right, due to chronic arachnoiditis, was made. From that time the child made continuous progress, and she was discharged as cured on May 23. She was seen about a year and a half afterward, had no complaints and seemed perfectly normal except that there was still some nystagmus when she looked to the right; there was no bulging over the decompression, and the ear was dry.

The internal hydrocephalus here perfectly simulated an abscess of the brain, particularly in the later phases of the child's illness. My diagnosis of an abscess of the temporal lobe rather than cerebellar abscess was based on the fact that the meningeal inflammation shown by the mastoid operation was limited to the middle fossa; the nystagmus I considered to be of a type originating in the brain stem and probably due to pressure. A case of hydrocephalus of the lateral ventricle in which the history and findings were identical except that there were also adiadokoknesis, asynergy and hypermetria has been reported by Gritti.[10] Williams[11] cited a case from the Mayo Foundation in which the findings were similar; exploration for cerebellar abscess was without result, and the patient later was found to have ventricular hydrocephalus on the side of the involved ear.

Abscess of the temporal lobe may be simulated also by an abnormal collection of cerebrospinal fluid between the dura of the middle fossa and the cortex. Such a collection may either communicate with the rest of the subarachnoid space or be encysted, so that lumbar puncture neither suggests the condition nor suffices for a cure. The spinal fluid in the communicating type is generally clear but often shows a moderate increase either in lymphocytes or in neutrophils. The spinal fluid in the case of ventricular hydrocephalus associated with infection of the ear may vary from a clear fluid to one with a cell count in the hundreds. Bruzzi,[12] of Naples, recently reported an interesting case of cystic arachnoiditis of the optochiasmic cistern at the extreme end of the middle fossa which followed severe mastoiditis. There were high grade papilledema and marked contraction of the visual fields inferiorly, and spinal fluid obtained by lumbar puncture was normal. The patient was cured by the passage of a needle along the superior margin of the petrous bone and evacuation of the fluid.

Cerebellar abscess may be closely simulated by a collection of fluid in the cisterna pontis lateralis. Some time ago I published a case which

10. Gritti, P.: Idrocefalia ventricolare otogena, Boll. d. mal. d. orecchio, d. gola, d. naso **55**:287, 1937.

11. Williams, H. L.: Otitic Hydrocephalus, Arch. Otolaryng. **25**:632 (June) 1937.

12. Bruzzi, B.: Un caso di meningite chiasmatica otogena, Boll. d. mal. d. orecchio, d. gola, d. naso **55**:241, 1937.

I believe was of this type.[13]. Here also, if a communication exists throughout the subarachnoid space the condition will clear up spontaneously or after a lumbar puncture, while if the fluid is encysted opening the dura is necessary.

Generalized Hypertension with Symptoms Referable to the Posterior Fossa.—The third type of arachnoiditis is the original "meningitis serosa" of Quincke,[14] later made known to otologists by Bárány.[15] The symptoms are well known: an acute onset with or without mild infection of the middle ear, the symptoms being headache, vertigo, nystagmus, cerebellar signs and increased intracranial pressure. When the condition is more severe, papilledema occurs. Spinal puncture, which generally shows a clear or almost clear fluid under great pressure, often suffices for a cure. In cases of this type thickening of the arachnoid and at times a gelatinous appearance of the brain have been seen at operation.[16]

PROBLEMS CONNECTED WITH ARACHNOIDITIS

The Nature of the Underlying Process.—An abnormal collection of fluid of the type under consideration within the cranial cavity may, as previously indicated, take the form of a cyst between the arachnoid and the brain, of internal hydrocephalus or of generalized intracranial hypertension with free communication throughout the subarachnoid space. For practical purposes certain terms, such as pseudoabscess or pseudotumor, are of value because they possess clinical connotation, but they correspond only in a limited sense to any variety of arachnoiditis. Thus while cystic arachnoiditis implies a slower process, which simulates tumor for the neurosurgeon, it may just as readily be mistaken for an abscess of the brain if it occurs during an active suppuration of the ear; the same may be said of the other two forms mentioned. Much has been attempted by various writers in the way of classification of the different forms of abnormal collection of cerebrospinal fluid under headings which will at once express a pathologic condition and a clinical concept. A survey of the literature serves to indicate that no hard and fast division can be made on this basis. The serous meningitis of the neurologist, with severe symptoms referable to the posterior fossa, shows in the hands of the neurosurgeon definite changes of the arachnoid or brain. Otitic hydrocephalus of the ventricles may be followed

13. Shapiro, S. L.: Pseudocerebellar Abscess, Arch. Otolaryng. **25**:17 (Jan.) 1937.

14. Quincke, H.: Ueber Meningitis serosa und verwandte Zustände, Deutsche Ztschr. f. Nervenh. **9**:149, 1896.

15. Bárány, R.: Syndrome of Bárány, Tr. Internat. Cong. Med. **14**:571, 1913.

16. Bourgeois and others.[7] Davis and Haven.[8]

by suppurative meningitis.[17] The spinal fluid is not always clear or limited to one type of cellular content. One fact, however, appears to be established: Whether the condition was acute or chronic, when there has been an opportunity either by operation or by necropsy examination to observe the brain the chief changes have been noted in the arachnoid. This seems to indicate that the arachnoid bears the brunt of the defense of the brain against the toxin or organism which is the causative factor.

A few words may be said about the source of the fluid in arachnoiditis. The accepted normal mechanism has been mentioned; it is well known, however, from both clinical and experimental evidence that under pathologic conditions the vessels of the pia-arachnoid may be stimulated to the production of cerebrospinal fluid. This fluid, however, differs from the normal fluid by showing an increased cell count and globulin reaction.[18] This fact may serve to differentiate cases in which increased intracranial pressure is due to hypersecretion; if the fluid is clear the condition would be considered the result of some disturbance of the choroid plexus, while if there is an increased cell count it would be regarded as due in part to an exudate from the pia-arachnoid. It is to be noted in this connection that in cases of ventricular hydrocephalus the fluid taken from the ventricle has approximated the normal fluid in appearance and content.

Diagnosis and Management.—Attempts have been made by Symonds,[3] Bourgeois [7] and others to lay down rules for differentiating arachnoiditis from tumor or abscess of the brain. An examination of these rules affords no certain guide for this purpose, and it is my opinion that the diagnosis of the condition cannot be made even by inference except in cases of a mild form in which a cure occurs spontaneously or after lumbar puncture. The condition may be suspected when the symptoms of increased intracranial pressure are out of proportion to the focal signs in the presence of an actual or previous aural infection, particularly in the case of a pseudotumor of the cerebellopontile angle when the involvement of the eighth nerve is not in accord with the other findings suggestive of tumor.

The role of the otologist in cases of arachnoiditis of the posterior or the middle fossa may, nevertheless, be of considerable importance. With the chronic variety the vestibular findings often, as in the case cited, furnish the first objective sign of an intracranial lesion; with an

17. Garland, H. G., and Seed, G. S.: Otitic Hydrocephalus, Lancet **2**:751, 1933.

18. Guttmann, L.: Physiologie und Pathologie der Liquormechanik und Liquordynamik, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 7, pt. 2. Plaut.[6]

acute condition associated with infection of the ear the track of the infection as shown by the previous findings in the ear and the appearance of the dura through the mastoid wound may give a clue as to which fossa should be explored.

The management is self evident. When an abnormal collection of fluid is suspected as the cause of the symptoms, exploration of the dura is indicated if after a reasonable period there has been no improvement. The older literature [19] and even some of the later reports record a number of fatalities, apparently from continued intracranial pressure alone, as well as several cases of optic atrophy following the choked disk, which is nearly always present; this indicates that in cases of severe arachnoiditis watchful waiting is a virtue which can be carried to excess.

---

19. Warrington, W. B.: Intracranial Serous Effusions of Inflammatory Origin, Quart. J. Med. **7**:93, 1914.

THE NASOGENITAL RELATIONSHIP

SAMUEL ROSEN, M.D.
NEW YORK

Since the time of Hippocrates medical literature has contained many notes concerning the intimate relationship existing between the nose and the generative system. The first extensive and accurate observations of the phenomenon were presented by John Noland Mackenzie,[1] a Baltimore surgeon, in 1884. Three years later Fliess [2] noted similar nasogenital relationships. He developed a therapeutic technic of cocainization of the "genital spots" in the nose for the alleviation of menstrual dysfunctions. In the United States, Brettauer [3] in 1911 reported good results in a large series of cases of dysmenorrhea in which the condition was treated by cocainization and cauterization of the nose. Seifert [4] made a critical study of the literature and cited 300 publications concerning the nasogenital relationship. For some time after these studies appeared, interest in this phenomenon dwindled; recently, however, it has been revived.[5]

Mortimer and his associates from the Montreal General Hospital investigated the nasal mucosa of 60 pregnant women. Their findings

---

From the Mount Sinai Hospital.

Candidate's thesis for admission to the American Laryngological, Rhinological and Otological Society, Inc.

1. Mackenzie, J. N.: Irritation of the Sexual Apparatus as an Etiological Factor in the Production of Nasal Disease, Am. J. M. Sc. **87**:360, 1884; The Physiological and Pathological Relations Between the Nose and the Sexual Apparatus of Man, Bull. Johns Hopkins Hosp. **9**:10, 1898.

2. Fliess, W.: Ueber den ursächlichen Zusammenhang von Nase und Geschlechtsorgan, zugleich ein Beitrag zur Nervenphysiologie, Halle, Carl Marhold, 1902.

3. Brettauer, J.: Dysmenorrhea Relieved by Nasal Treatment, Am. J. Obst. **64**:214, 1911.

4. Seifert, E. H. E.: Kritische Studie zur Lehre vom Zusammenhang zwischen Nase und Geschlechtsorganen, Würzburg, Curt Kabitzsch, 1912.

5. (*a*) Mortimer, H.; Wright, R. P.; Bachman, C., and Collip, J. B.: Effect of Oestrogenic Hormone Administration on Nasal Mucous Membrane of the Monkey (Macaca Mulatta), Proc. Soc. Exper. Biol. & Med. **34**:535, 1936. (*b*) Mortimer, H.; Wright, R. P., and Collip, J. B.: Effect of Administration of Estrogenic Hormones on the Nasal Mucosa of Monkey (Macaca Mulatta), Canad. M. A. J. **35**:503, 1936; (*c*) Effect of Estrogenic Hormones on Nasal Mucosa: Their Role in Naso-Sexual Relationship and Their Significance in Clinical Rhinology, ibid. **35**:615, 1936. (*d*) Rosen, S., and Shelesnyak, M. C.: Induction of Pseudopregnancy in the Rat by Silver Nitrate on the Nasal Mucosa, Proc. Soc. Exper. Biol. & Med. **36**:832, 1937.

revealed the nasal reaction of increased redness and swelling in the later stages of pregnancy. In 50 per cent of their cases there was epistaxis during pregnancy. Their report presented concise reviews of the salient studies of Mackenzie,[1] Endriss, Fliess,[2] Seifert [4] and Freund.

Manifestations of the nasogenital relationship may be considered from two aspects: first, the influence of genital functions on the nose, and second, the influence of the nasal status on genital function.

The observation of Mackenzie [1] that in a certain percentage of normal women there is hyperemia and swelling of the nasal mucosa during menstruation has been amply confirmed. Periodic epistaxis has been reported as vicarious menstruation. Many reports have been made associating nasal "stuffiness and sneezing" with acts of copulation (Endriss, Morell, Mackenzie) and sexual excitation. In the presence of ozena there is frequently found at the time of menstruation an increase in size and vascularity of the nasal mucosa, with alleviation of the nasal symptoms. These are all instances of a response of the nasal physiologic function to states of genital function.

From the laboratory have come certain limited evidences of the influence of the genital status on the nasal condition. Gonadectomy is followed by degenerative changes in the nasal mucosa. Mortimer, Wright and Collip [5] demonstrated hyperemia and hypertrophy of the nasal mucosa of castrated monkeys which received injections of estrogens.

On the other hand, the influence of the nasal state on genital behavior has a lesser array of reported instances. Mackenzie [1] cited a remarkable case of Heschel (*Wiener Zeitschrift,* March 1861) in which genital hypoplasia was associated with absence of olfactory bulbs and mucosal nerves. Most notable is the effect on dysmenorrhea of nasal treatment, as reported by Fliess [2] and Brettauer.[3]

Recently in the laboratory I have demonstrated genital response to nasal treatment. Some workers concerned themselves chiefly with changes in the gonads after turbinectomy (Koblanck and Roeder, Nemours [6]).

In an effort to study the mechanism of nasogenital relationship, experiments were carried out in the laboratory of the Mount Sinai Hospital. The rat was chosen as the test animal because of its standardized sexual cycle.

It may be of advantage briefly to examine the reproductive cycle of lower mammals. At certain definite times, when the female shows sexual desire and accepts the male, the animal is said to be in "heat"

6. (*a*) Koblanck and Roeder, H.: Tierversuche über Beeinflussung des sexual Systems durch nasale Eingriffe, Berl. klin. Wchnschr. **49**:1893, 1912. (*b*) Nemours, P. R.: Effect of Experimental Bilateral Turbinectomy on the Development of the Testes in the Rabbit, Arch. Otolaryng. **22**:626 (Nov.) 1935.

or in estrus. During the interval between two successive periods of estrus, the genital system undergoes a cycle of changes in form and function, designated the "estrus cycle." The estrus cycle is composed of two phases: a follicular phase of proestrus and estrus, associated with the growth of the graafian follicles of the ovary, and a luteal phase of diestrus, associated with the development of corpora lutea from ovulated follicles.

The principal changes which take place in the reproductive organs of the rat are the following: During proestrus a number of graafian follicles begin to mature rapidly, fill with liquor folliculi and move to the surface of the ovary. The vaginal mucosa is hypertrophied and demonstrates definite cyclic modifications, particularly by its epithelial cells. These cells, which are easily shed, can be examined microscopically in a smear of the vaginal contents. Study of these vaginal smears affords a convenient and expedient method for detecting the exact stage of the estrus cycle. The original work on the estrus cycle of the rat and on its associated phenomena was done by Long and Evans [7] and was published in 1922. The vaginal smear of the rat during the proestrus period shows oval nucleated epithelial cells.

During estrus the follicles reach their full growth and maturation, and ovulation ensues. The vaginal mucosa becomes exceedingly cornified, and smears consist wholly of desquamated scales and non-nucleated cornified cells.

The course subsequent to estrus depends on whether fertilization occurs. If so, pregnancy follows; if fertilization does not occur, diestrus follows. The uterine wall shows some thickening and some glandular hypertrophy. The vaginal smears present leukocytes. If a nonfertile or so-called sterile coition takes place the resultant condition of the female is termed pseudopregnancy. The corpora lutea of the ovary persist; the uterus shows the progravid type of endometrium; the vaginal smears are of the diestrual or leukocytic type. The pseudopregnant uterus possesses a definite reactivity to trauma, so that a properly timed traumatization produces a transitory decidual cell tumor, a deciduoma. The fact that this growth can be elicited signifies the existence of a pseudopregnant uterus—a uterus that has responded initially to estrin (from the follicles) and subsequently to progestin (from the corpora lutea).

The normal length of the estrus cycle in the rat is four days. Proestrus lasts about twelve hours; estrus, about thirty-six hours, and diestrus, forty-eight hours. The cycle of pseudopregnancy is from eight to twenty-two days in duration, averaging fourteen days.

7. Long, J. A., and Evans, H. M.: The Oestrus Cycle in the Rat and Its Associated Phenomenon, University of California Memoirs, Berkeley, University of California Press, 1922, vol. 6.

In the spring of 1936 I began a series of experiments in treating the nasal regions of rats. The nature of the estrus cycle after treatment was observed. In the course of the past eighteen months 400 female albino rats were treated and observed.

Adult female albino rats from an inbred colony were used. They were amply housed and were completely isolated from males. The colony diet consisted of hospital table scraps supplemented with grain, greens, powdered milk, pablum and cod liver oil. The regularity and rhythmicity of the estrus cycles were determined from daily vaginal smears. Throughout the entire study only animals with four to five day cycles were used. Treatment was begun only after the observation of four to six consecutive regular periods. All treatments were given while the animals were under light ether anesthesia.

Treatment consisted of application of various solutions to the nasal mucosa either with small cotton applicators or, more generally, by irrigation with a catheter-tipped syringe.

Treatments were made at different phases of the estrus cycle. One group of animals was treated once daily on the days of proestrus and estrus of two successive cycles. A second group was treated once daily for six days, which included two days of diestrus and the four days of the succeeding cycle. A third group was treated during this same six day period, but twice daily.

The details of this treatment are published elsewhere. To summarize briefly, these are the essential facts. In the first group of experiments 186 animals were given various concentrations of silver nitrate solutions. Of these 186 rats, 127 received intranasal treatment. The response of the genital system was a prolonged leukocytic phase. The vaginal smears of 54 per cent of the animals were typical of pseudopregnancy, and 3 traumatized uteri produced deciduomas. It was concluded from this study that silver nitrate given intranasally provoked pseudopregnancy in the rat.

As the action of silver nitrate can range from astringent to irritant, the nature of the nasal tissue reaction evoking the pseudopregnancy could not be ascertained from studies with silver nitrate. In the next groups of experiments I attempted to simulate specific actions of the silver salt, namely, irritant action and depressant action.

Thirty-nine animals were given oil of mustard, some by application, others by irrigation. Nineteen animals showed an alteration of the regularity of the cycles; most of the irregularity was in reference to the follicular phase of the cycle, since the vaginal smears revealed additional days during which the cells were cornified.

The depressants of the nasal tissue were of two types, protein precipitants and anesthetics. Of a limited group of 20 animals which received protein precipitants, either 10 per cent tannic acid solution or 10 per cent trichloracetic acid solution, 7 showed prolonged leukocytic phases after treatment.

The anesthetic series was most extensive, comprising 138 rats. All animals received nasal irrigations of 2 per cent nupercaine solution (Ciba) in 0.9 per cent saline solution. The time of application of the anesthetic in reference to the estrus cycle varied. Fifteen rats received single daily treatments during proestrus and estrus of two successive periods; 36 animals received single daily treatments for six days, 2 diestrus and 1 proestrus, 1 estrus and 2 diestrus; and 63 received six day treatments, but twice daily. The anesthetic was administered to 24 controls in the oral cavity.

Exactly half the 114 experimentally treated rats showed prolonged leukocytic phases. The prolonged cycles were the same length as pseudopregnancy, namely, nine to twenty-two days. Although 50 per cent of the entire group reacted to treatment, the reaction was not uniformly distributed through the series. For example, one series, in which 15 animals were treated on days of estrus and proestrus of 2 consecutive cycles, showed response in only 3 instances. Another series of 25 rats treated twice daily showed 20 positive reactions. Control animals showed negative reactions with the exception of 1 rat which had a nine day cycle.

The length of the prolonged cycle approximated the length of pseudopregnancy, and the ovaries showed luteinization. Nevertheless (as a test of the pseudopregnant state of the endometrium), a group of animals was operated on and the uteri traumatized in an effort to provide decidual response. Sixteen animals were laparotomized on the fourth day of the leukocytic phase of the prolonged cycle. A silk thread was passed through the lumen of the uterus, and a loose loop was tied. Four days later the uteri were examined for decidual growths. Eleven showed decidual tumor masses. This is regarded as definite evidence of the existence of a progestational type of endometrium.

COMMENT

The mechanism of the nasogenital relationship, especially with reference to the influence of the nasal sphere on gonadal behavior, has not been investigated. The elicitation of a response of the gonads by manipulations of the nasal mucosa presented a demonstration of the relationship, but on account of the variant nature of the action of silver nitrate, it offered no clue to the mechanism. I am pursuing the investigation and attempting to ascertain the type of nasal tissue reaction which leads to pseudopregnancy. My experiments have revealed that local depression of the nervous system is certainly correlated in a large percentage of cases with modified estrus cycles; but the problem is by no means solved, and to say that I am approaching a solution would betray much optimism. Nevertheless, it is true that the nasogenital relationship has been demonstrated and the involvement of the nervous elements of the nasal mucosa has been evidenced. Investigation of the pathways and of the type of gonadal response are now in progress, and I hope that in good time they will lead me to knowledge and understanding of the complexities of the nasogenital relationship.

MORPHOLOGIC AND ROENTGENOLOGIC ASPECTS OF THE TEMPORAL BONE

STUDY OF 536 BONES WITH SPECIAL REFERENCE TO PNEUMATIZATION

C. C. ROE JACKSON, M.D., D.Sc. (MED.)

CLEVELAND

The more one studies the temporal bone with its vital parts and associations, the more one realizes that this study still offers a great deal which is unknown. Various great anatomists, physiologists, surgeons and clinicians have their names, as a result of their work, attached to various parts of the bone, theories about it and clinical conditions affecting it.[1] Herman Ferdinand von Helmholtz (1821-1894) in 1863 published his theory of hearing, "Tone Sensations"; Julius Richard Ewald (1856-1921) described the semicircular canals (semicircles of Ewald) and their part in equilibration; Henle's spine bears the name of Jacob Henle (1809-1885), who described it as a reliable guide in locating the antrum in surgical intervention in the mastoid region, and Sir William Macewen (1848-1924) methodically laid out the triangle (Macewen's triangle) as a guide in the mastoid operation. In addition, one can mention the depression of Wilde, the tubes of Eustachius, the hiatus of Fallopius, the space of Prussak, the membrane of Shrapnell, the organ of Corti and others.

One of the greatest contributions to knowledge of the anatomy and pathology of the temporal bone in the last quarter-century was that of Karl Wittmaack,[2] who in 1918 published his book entitled "Ueber die normale und die pathologische Pneumatization des Schläfenbeines," consisting of a text and an atlas. Working alone, Wittmaack took ten years

---

From the Department of Surgery, School of Medicine, Western Reserve University, and the University Hospitals.

Thesis submitted to the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Doctor of Medical Science (D.Sc. [Med.]) for graduate work in otolaryngology.

1. Effler, L. R.: The Eponyms of Anatomy, Toledo, Press of the McManus-Troup Company, 1935. Garrison, F. H.: History of Medicine, ed. 4, Philadelphia, W. B. Saunders Company, 1929.

2. Wittmaack, K.: Ueber die normale und die pathologische Pneumatisation des Schläfenbeines (einschliesslich ihrer Beziehungen zu den Mittelohrerkrankungen), Jena, Gustav Fischer, 1918.

to obtain his material, 300 temporal bones which were sectioned for microscopic study. Some bones were removed from cadavers unchecked by clinical observation during life. Others were recovered from actual patients. From these he drew his conclusions. It is nearly twenty years since he published this painstaking and thorough pathologic investigation, and no contradictory view of any scientific weight has appeared. This work gave the otologic world a new concept of the anatomicopathologic features commonly met in work with the gross temporal bone, which, after a lapse of ten years, has been the basis of further study during the past decade. One may justly conclude, then, that the theory of pneumatization advanced by Wittmaack is universally accepted today.

Wittmaack's [2] rich findings in following up the correlation between the character of the mucous membrane of the middle ear and pneumatization of the mastoid process, on the one hand, and between the diseases of the mucosa and arrest of pneumatization, on the other hand, constitute an almost complete working out of the normal and pathologic anatomy of the mucous membrane of the middle ear. He demonstrated the normal pneumatization extending completely throughout the mastoid process but did not include the extensions into the apical part of the pyramid. In the last decade these apical ramifications of pneumatization have been considered definite lethal factors in many of the cases in which fatal intracranial complications developed. As a result, studies have been conducted by many workers in various parts of the world, making otologists conscious of the anatomic background of this clinical entity so that they might cope with it more intelligently.

Hammar (cited by Keibel and Mall [3]) recognized three periods in the development of the middle ear. The period of formation is that of the primary tympanic cavity (when the embryo is 3 to 18.5 mm. in length); it consists of the dorsal prolongation of the first pharyngeal groove, the posterior tympanic groove and the portion of the impressio cochlearis which at first lies medial to these structures. The second period is the period of separation (ending when the embryo is 24 mm. in length). The third period is the one in which the tubotympanic canal is changed into the definitive tympanic cavity and the tuba auditiva. This period continues into postfetal life. G. L. Streeter [4] in 1907 stated that "the labyrinth of the 30 mm. embryo has practically completed its gross development." Hence the labyrinth has reached full development before the beginning of the period of transformation (third period) of Hammar.

---

3. Keibel, F., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1912, vol. 2, pp. 272-287.

4. Streeter, G. L.: On the Development of the Membranous Labyrinth and the Acoustic and Facial Nerves in the Human Embryo, Am. J. Anat. **6**:151, 1907.

According to Wittmaack,[2] there is first the formation of the cavum, the recessus and the antrum. At birth these are merely potential cavities, being filled almost entirely with myxomatous tissue covered by cuboidal epithelium, and a mere slit exists between the gelatinous mass and the tympanic membrane. Gradually the layer of myxomatous tissue becomes thinner, following the outlines of the cavities more closely, and the epithelium, in turn, lines the enlarging cavum, the recessus and finally the antrum. Normally the myxomatous tissue is finally represented by a thin subepithelial layer over the bone, and the cuboidal epithelium of the embryo changes to the epithelium of the adult. The mastoid process, rudimentary of course, consists of spongy bone (spongiosa) until the end of the first year of life.

The second stage Wittmaack has placed from the beginning of the second year to the fifth year of life. The epithelium from the antrum dips down into the marrow cavities in the immediate vicinity, and, as it dips down, the myxomatous tissue, its precursor, is resorbed until it becomes the thin submucous layer. Thus an air space is formed. The marrow cavities of the mastoid process, invaded by myxomatous tissue, which in turn is followed by epithelium, continue to exist until the end of the fourth or the beginning of the fifth year of life, when the larger air cells are completed. He names the third stage, which extends throughout life, the stage of interstitial pneumatization. It consists of the formation of smaller pneumatic spaces communicating with the larger ones, so that the cell nearest the cortex of the mastoid process must be the last to become a part of the system.

His conclusions regarding normal pneumatization, then, were the following:

1. The development of the pneumatic system of the mastoid process follows in its normal course far more definite laws than has heretofore been supposed.

2. The position of the solitary air spaces is brought about by regular and typical, recurring histologic changes.

3. When development is undisturbed the mastoid portion appears toward the end of the first year of life as a spongy structure (first period of development); from then to about the fifth year of life it appears as a mixed spongy, pneumatic structure (second period of development), and, beyond this, regular complete pneumatization takes place (third period of development).

4. Even under completely normal conditions the process of pneumatization never comes to a complete standstill; both interstitially and peripherally, single pneumatic cells are formed even up to advanced age.

5. The normal construction of the mastoid process in advanced age is characterized by extensive pneumatization, not by sclerosis.

6. Individual variations in the development of the pneumatic systems in later life (after the fifth year) make their appearance no longer in the histologic picture of the mastoid process, but only in the extension of the single peripheral processes of the pneumatic cells.

If under normal conditions it is possible for the mastoid process to become completely pneumatized, it is equally possible for the apical or carotid portion of the pyramid to become pneumatized. Thus, in the light of recent studies, it may be said that, unmolested by any irritation, the mucous membrane of the middle ear progresses to establish complete pneumatization of the whole pyramid, external and medial to the internal ear. Wittmaack explained pathologic pneumatization as anything which differs from complete pneumatization of the mastoid process. According to Aschoff, amniotic fluid, vernix caseosa or meconium may be aspirated before birth into the cavity of the middle ear; shortly after birth vomitus may be forced into it or inflammatory processes occur in it. Any of these accidents, separately or in combination, may cause in the mucous membrane inflammatory changes which arrest or retard pneumatization. If inflammation occurs during the first months of life, when the mucous membrane still has its high subepithelial cushion, this high layer furnishes resistance to the extension of epithelium into the marrow spaces now prepared for pneumatization. The periosteum adjacent to the dura and the outer periosteum continue to produce new bone, and, since pneumatization is arrested, there results an almost completely sclerosed mastoid process.

His conclusions regarding pathologic pneumatization were the following:

1. The deviations from the normal structure of the mastoid process depend, without exception, on typical arrest or destruction of the process of pneumatization, resulting from changes in the character of the mucous membrane in the first and the second year of life.

2. The variations in the character of the mucous membrane fall into the fibrous and hyperplastic types.

3. Hyperplasia of the mucous membrane develops because of a latent, slowly creeping, plastic inflammatory process.

4. Fibrosis develops in consequence of acute inflammatory processes.

5. According to the intensity of the changes in the mucous membrane, the arrest of the process of pneumatization is either partial or complete.

6. Each form of arrest presents a typical picture in the structure of the mastoid process: (*a*) With complete arrest of pneumatization there goes hyperplasia or fibrosis of the mucous membrane. (*b*) With partial arrest of pneumatization there is also hyperplasia of varying degree and fibrosis of the mucous membrane retarding pneumatization.

7. Association of hyperplasia with superficial fibrous changes in the mucosa occurs rather frequently and leads to malformations of structure in which sometimes the one and sometimes the other component predominates.

8. The forward position of the lateral sinus occurs only in pathologic pneumatization and to a marked degree only in the most severe disturbances of pneumatization. Its development is otherwise dependent on the structure of the bone of the mastoid process.

9. Both persistence and unusual breadth of the fissures are concomitants of pathologic pneumatization.

Since the internal ear at birth is already as large as it is in advanced age, its size can be considered a constant factor. Longitudinal extension of the petrous pyramid during growth, conforming to general expansion of the base of the skull, may possibly be influenced by race, sex, heredity or environment. Groth [5] stated that the absolute development of the mastoid process is entirely independent of pneumatization. Wittmaack taught that pneumatization of the mastoid process is exceedingly variable, so that indeed it seldom spreads to the limits of possible extension. If that is true of the mastoid process, it is even truer of the apex of the petrous pyramid. Nevertheless extensive pneumatization does occur, and, as will be seen, its relative frequency varies with age. When extensive pneumatization is present, a much greater area is open to infection than in the opposite type. Growth accompanied by normal pneumatization should produce a pyramid of greater volume than is produced when pneumatization is arrested. By gross examination of petrous pyramids one can note considerable difference in the apparent volume, but roentgenographic examination is necessary to determine without destroying the bone the presence or absence of pneumatization.

In 1934 Myerson, Rubin and Gilbert [6] reported their results from an anatomic study of 200 temporal bones from 100 autopsies. Seventy per cent of these specimens showed some degree of pneumatization, and 30 per cent were sclerotic; of the pneumatized ones, 11 per cent showed pneumatization extending into the apex of the pyramid; 36 specimens from children up to 7½ years of age showed no pneumatic petrous apexes, and 4 of the 100 skulls presented pneumatization of one petrous apex but not of the other. These authors also maintained that there is a greater incidence of pneumatization of the pyramid in the Negro than in the white race. In the same year Hagens [7] reported his study of 50 temporal bones. He observed pneumatic spaces in the apex of the pyramid in 34 per cent. Incidentally, he gives the distance between the cochlea and the internal carotid artery, just below the level of the tegmen tympani, as ranging from 4 to 10 mm., the average being 6.2 mm. No record is made of age, sex or race.

Just where the nodal areas for apical pneumatization may be is as yet an unsettled question. The pneumatic spaces which extend around, under, behind and above the labyrinth primarily begin from the antrum or the posterior inferior portion of the tympanic cavity. It has been

5. Groth, W.: Der Warzenforsatz des Menschen als Knochenleiste am Schädel und sein inneres Gefüge, Anat. Anz. **85**:249, 1938.

6. Myerson, M. C.; Rubin, H. W., and Gilbert, J. G.: Anatomic Studies of the Petrous Portion of the Temporal Bone, Arch. Otolaryng. **20**:195 (Aug.) 1934.

7. Hagens, E. W.: Anatomy and Pathology of the Petrous Bone Based on a Study of Fifty Temporal Bones, Arch. Otolaryng. **19**:556 (May) 1934.

asserted that pneumatization of the apical or of the carotid portion of the pyramid may begin near the tympanic orifice of the eustachian tube and extend along the wall of the canal of the internal carotid artery. There is no argument against this assertion, providing the bone marrow is present in this area and the mucous membrane of the middle ear maintains its invasive property. If one could assume that the mucous membrane of the anterior part of the cavity of the middle ear might escape the retarding influence, which would instead affect the posterior part, a pneumatic apex might be accompanied by arrest of pneumatization in the mastoid process. So far as is known, this does not occur, probably because the mucous membrane of the middle ear responds as a unit.

MATERIAL

Dr. T. Wingate Todd, Henry Willson Payne professor of anatomy, Western Reserve University Medical School, over a period of years has collected and preserved over 3,000 skeletons, and it was from this collection of material that a suitable selection of specimens was made for this study. The material remains in its original state, so that future students may use it, together with whatever additional material is subsequently collected, for further studies. Throughout the study Dr. Todd has given free access to the material, the laboratory assistants' aid, the apparatus, the records and the original work of assessment.

Even in a collection of over 3,000 skeletons there are some periods in the life span for which there is a dearth of material. Abundant material exists for the periods before the age of 1 year and after the age of 21. The material for the interval from 1 to 20 years, particularly from 2 to 15, at present leaves much to be desired, despite the fact that the Hamann Museum contains many times as much material for the youthful period as is available for study in any other center. The sparsity of material suitable for the study of this period may be explained by the fact that there is greater sentiment attached to the final disposal of the bodies of children. All specimens representing the ages above 20 have been prepared and preserved as dry material. A considerable number from subjects from 16 to 20 years old also were dry specimens, but most of those from subjects below 20 and all those from persons below 16 were wet preparations. A goodly number of skulls were unsuitable for this study because postmortem examinations had included the middle ear and accordingly that portion of the pyramid had been damaged or removed. The wet specimens were more difficult to handle in mensural and roentgen study but were of fundamental value in establishing standards.

The instruments used to make the several measurements on the skulls and temporal bones were the Western Reserve spreading calipers, special small outside spreading calipers and a rule. They are illustrated in figure 1. Before the actual study was begun, however, some experience was obtained in taking and reading the various measurements under the supervision of Dr. W. M. Krogman, associate professor of anatomy and physical anthropology. The roentgen study was conducted in the laboratory of anatomy, the procedure being that each bone was placed in position while Miss Dorothy Lane, research assistant in anatomy, made the exposures. Miss Lane also arranged the identification numbers and developed

the films. The anatomic records were consulted to ascertain data about chronologic age, sex, race, cause of death and maturation age (Todd,[8] Hill [9]). Free access to the anatomic library, the university library and the Cleveland Medical Library was given.

PROCEDURE

In collaboration with Miss W. Kuenzel, specimens in several positions, including stereoscopic setups of the temporal bone, were arranged and examined roentgenologically. When sample roentgenograms in all these positions had been obtained and studied carefully, the decision was finally made to orient the skull as in figure 2 *A* and *B* and make a nonstereoscopic roentgenographic record. For anatomic study this position was found to be the best, and it was used consistently throughout the study. In cases of doubt, stereoscopic roentgenograms were substituted. All the skulls except a few, and those mostly of the very young, were sectioned sagittally in the midline. Accordingly, taking a half skull, setting the base on the x-ray plate and tipping the vertex off center 40 degrees, with the posterior fossa raised by support under the occipital bone, and centering the x-ray tube over the arcuate eminence, one gets the position used in this study. The skulls when placed in this position were tilted off center enough to cast the shadow of the vertex beyond the pneumatized area. For this purpose an average tilt of 40 degrees was required—in some skulls more, in others less. All skulls

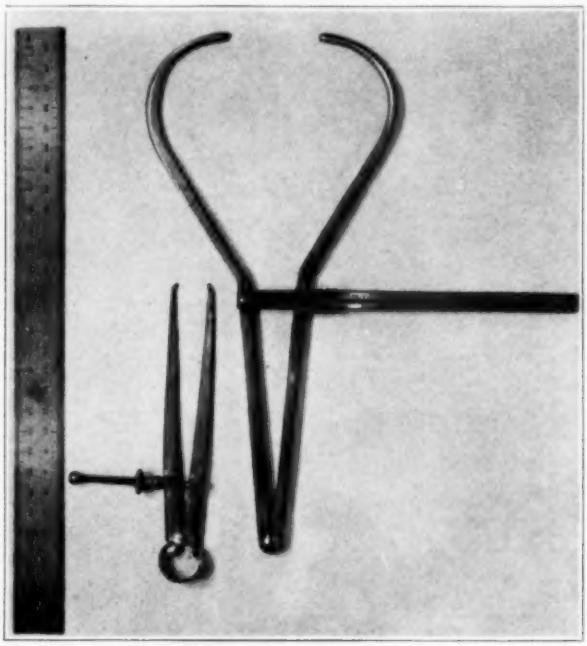


Fig. 1.—Instruments used in taking measurements.

8. Todd, T. W.: Atlas of Skeletal Maturation, St. Louis, C. V. Mosby Company, 1937, pt. 1.

9. Hill, A. H.: Fetal Age Assessment, to be published.

were held in position by plasticine during the roentgen exposure. Figure 2*C* illustrates the position of choice. Except in a few instances, this position and a uniform density, so far as was possible, were maintained throughout the study, in order to show most clearly the structure under consideration. The right and left temporal bones received the same exposure and appear on the same film, so that there was an identical procedure for each bone of the same skull. The roentgenograms were numbered to correspond to the museum's index number for that skeleton, and the right and left sides were indicated as R and L.

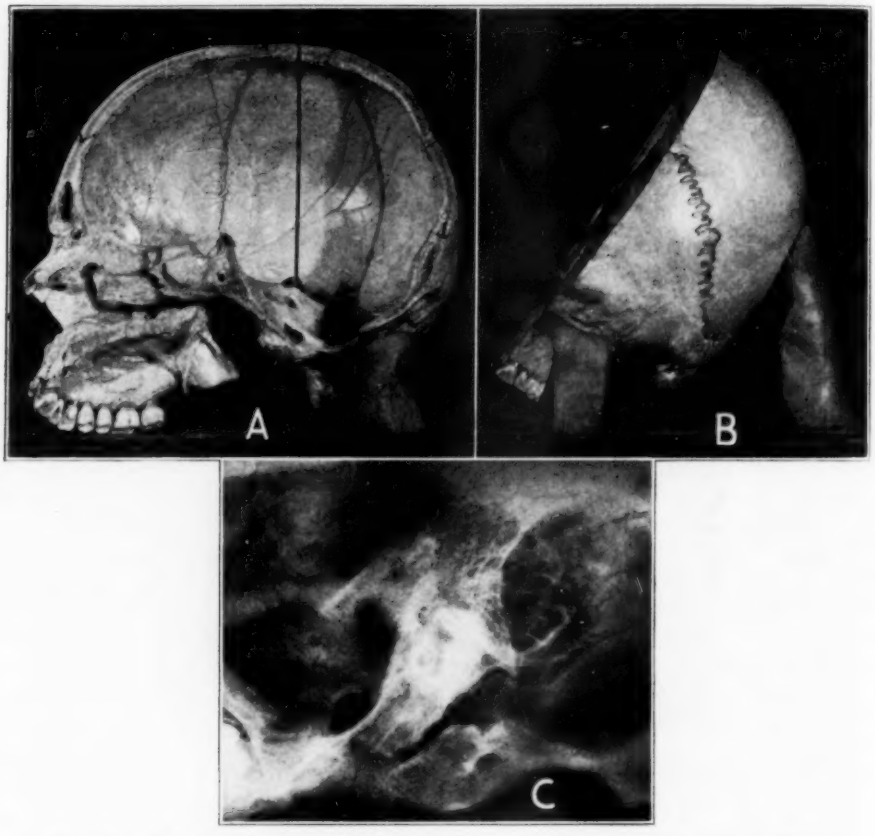


Fig. 2.—*A*, roentgenogram in lateral view. The course of the x-ray beam is indicated by the pointer perpendicular to the x-ray plate and centered over the arcuate eminence. *B*, roentgenogram in posterior view. *C*, typical roentgenogram.

As a connecting link between the dried specimens and the living, a series of 20 adult temporal bones, varying in age, were examined before maceration and, as soon as dried, after maceration. From this series there was found to be no difference between the specimens in the green state and as dried material, except for greater clarity in outline of the several bony processes, foramens and fissures in the dried specimens. Air cells detected in the dried specimens were easily seen also in the films of the unmacerated specimens. The marrow cell, precursor of the air cell, could not be detected in the specimens from adults, but in those from

very young persons there appeared an area of differentiation, which in the process of undisturbed growth became replaced by air cells. Each film includes the basilar portion of the occipital bone and the body of the sphenoid, so that a rapid determination could be made as to whether or not the apex was diploic or contained air cells.

The anatomic mensural study of each temporal bone was conducted along lines which appear to be of academic value only but which may possibly have future significance in surgical intervention on the petrous pyramid of the bone. Landmarks are of fundamental value in operative work, especially on the temporal bone. Accordingly, some of the landmarks used in this study have been in use since surgical intervention in the mastoid region began, and some may never be of practical use owing to their inaccessibility. However, it is of academic interest to ascertain an average or standard. The various factors of development and disease concerned in the pneumatization of the temporal bone are believed to be definitely concerned in altering the distances which were measured. It can be shown that certain factors produce certain alterations in landmarks, and these factors can be detected first roentgenologically and second clinically. Thereby a forecast can be made as to the ease or difficulty of surgical procedure.

To consider the interrelation of landmarks one may take, first, (A)[10] the distance between the depression of Wilde and the arcuate eminence, i. e., between *1* and *2*, figure 3 *A*, *B* and *C*. In recording this measurement a common understanding must be established, namely, that the spreading calipers do not go to the depth of the depression, but the measurement represents the distance from the depression of Wilde to the anterior vertical semicircular canal. This anterior vertical semicircular canal is a landmark in certain technics of surgical approach to the apex of the pyramid. The second measurement (B) is the distance from the arcuate eminence to the medial tip of the apex, i. e., between *2* and *3*, figure 3 *B*. The third measurement (C) represents the thickness of the roof of the internal auditory canal, i. e., the distance between *4* and *5*, figure 3 *B*. A small measurement here makes it unlikely that cells are present within this plate of bone; a relatively great measurement, on the other hand, suggests, though it does not determine, the presence of pneumatization. Indeed, it may be by this route that pneumatization extends from the mastoid portion to the apex of the pyramid.

The fourth measurement (D) represents the minimal distance between the posterior wall of the canal of the internal carotid artery and the posterior surface of the pyramid, at approximately the middle of the apex, i. e., halfway between the turn of the carotid artery and the tip of the apex. This is the distance between *6* and *7*, figures *3 B*, *C* and *D*. The measurement, if small, would indicate in general a small volume, but would not exclude the potentiality of cellular development over or above the carotid canal. However, when the measurements are of greater caliber, there exists greater area or volume for the presence of pneumatic cells. The next measurement (E) records the height of the posterior aspect of the apical (carotid) portion of the pyramid approximately halfway between the internal auditory meatus and the medial tip of the pyramid, i. e., the distance between *8* and *9*, figure *3 B*. This measurement gives one some conception of the relative height of the carotid portion of the pyramid, so far as is possible for a tapering mass of bone. The minimal distance (F) between the hiatus Fallopii and the posterior superior edge of the pyramid (at right angles to the latter), i. e., between *10* and *11*, figure 3 *C*, for the most part is the distance between the medial wall of the superior part of the tympanic cavity and the posterior surface of the pyramid.

10. The data in the tables are designated by the letters used in this enumeration.

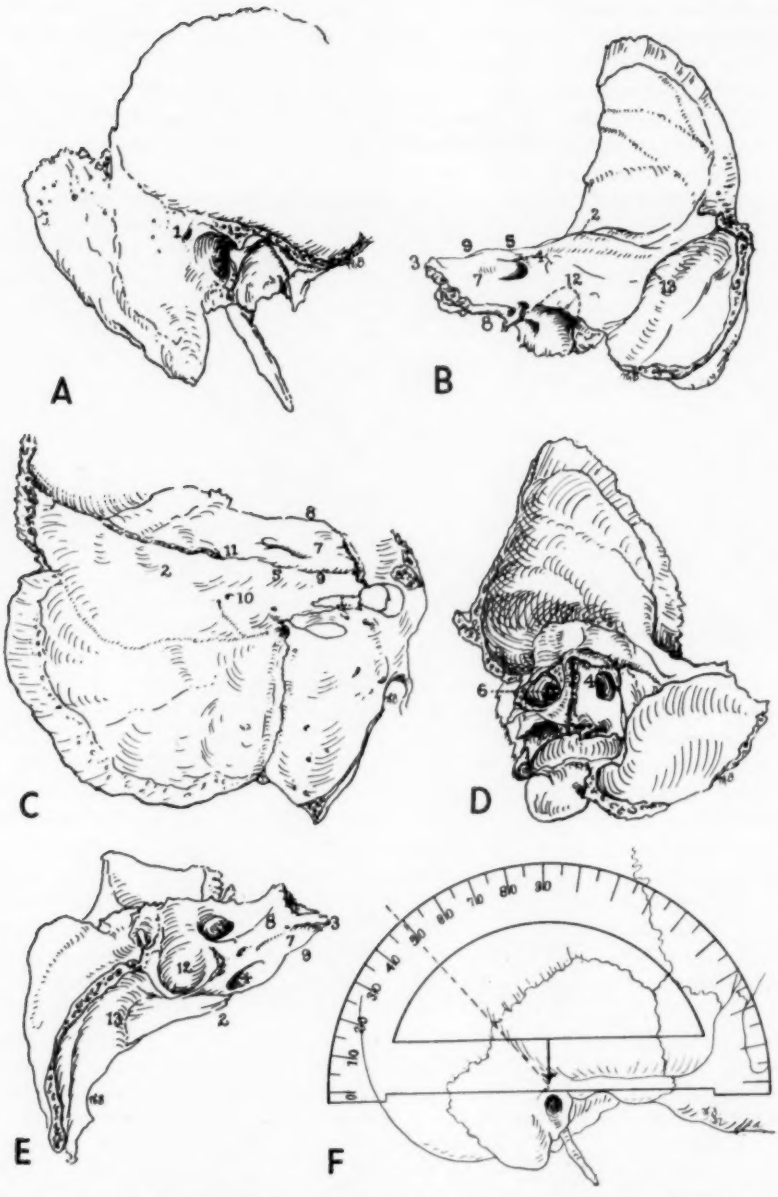


Fig. 3.—*A*, right temporal bone (zygomatic arch removed), view from without. *B*, right temporal bone, view from within and behind. *C*, right temporal bone, view from above. *D*, right temporal bone, view of the apex. *E*, right temporal bone, view from below. *F*, protractor placed over a reduced temporal bone, measuring a 50 degree angle of deviation of the posterior root of the zygoma from the plane of the zygomatic arch.

It is of great significance to record the minimal distance (G) between the highest part of the jugular fossa on the under surface of the temporal bone and the superior aspect of the arcuate eminence (anterior vertical semicircular canal), i. e., the distance between *12* and *2*, figure *3 B* and *E*. The greater the distance the more the possibility of pneumatization above or below the labyrinth, and air cells may even occur in both locations in the same bone. Notations (K) have been made as to the relative position of the jugular fossa, *12* in figure 3 *B*, i. e., high, medium or low. The higher its position, the thinner will be the jugular-tympanic wall. This wall may even show a dehiscence, which would potentially account for primary thrombosis of the jugular bulb from mild otitis media or severe bleeding following paracentesis when the jugular bulb is accidently incised.

The eighth measurement (H) gives the distance from the arcuate eminence to the anterior margin of the depression made by the swing of the lateral sinus, roughly at right angles to the tangent of this margin, i. e., between *2* and *13*, figure 3 *B*. This measurement is significant in surgical intervention on the temporal bone; it gives some idea of the size of Trautman's triangle and also of the extent of this area, which may or may not be pneumatized. The next measurement (I), not possible to record for the specimens from the youngest children, represents the greatest length and breadth of the bony dehiscence overlying the internal carotid artery and is shown by *i* in figure 3 *C*. Naturally the presence of a large dehiscence excludes the possibility of pneumatic spaces superior to the canal of the carotid artery.

The bony eminence or ridge which forms the continuation of the posterior root of the zygoma is alleged to have significance in surgical intervention in the mastoid region; if it continues in the same plane as the zygomatic arch the lateral sinus is likely to be some distance from the posterior wall of the canal. As opposed to this, the greater the angle it forms in turning up from the plane of the zygomatic arch the closer the lateral sinus is thought to be to the posterior wall of the canal. Accordingly, since it is comparatively easy to measure this angle (J) (fig. 3 *F*), it can be compared with the minimal distance (L) between the sigmoid part of the lateral sinus and the depression of Wilde, i. e., the distance between *1* and *13*, figures 3 *A* and *B*.

PHASES OF DEVELOPMENT BASED ON SKELETAL AGE

In the process of building up the standard on which the body of this study has been based, certain difficulties have been encountered. As mentioned before, there are periods in the lower ages for which there is a scarcity of material suitable for the study, and in these a great variation in chronologic and maturation ages is apparent. The explanation for this fact lies in the influence of time, maturation and health on the skeleton. The effect of these factors is important, especially in childhood. Unless some distinction is made, age relationships are most confusing. First in importance in bringing about change in the skeletal pattern is maturation, the effect of which is plainly seen, but over which there is as yet no control. The phases designated as birth, early childhood (2 years), later childhood (to 16 years) and adulthood are all evidence of the effect of maturation.

If one were to examine any chance series of skulls, a wide discrepancy between children of the same age would become evident. This

has been considered a natural variation. When, however, it is remembered that all skulls of dead children come from those who have been stricken with ill health, more or less prolonged, a reason for this variation becomes apparent. Chronically sick children are retarded in maturation, and the part which is undergoing the greatest change is definitely the most affected ("Atlas of Skeletal Maturation" [8]). In infancy and early childhood, retardation is most perceptible in the face and skull. It has been seen what great and rapid changes in the petrous bone are characteristic of this stage of life; therefore, a discrepancy roughly measured by contrast can be expected.

Now the stage of maturation of these children has been assessed by the methods described in the Atlas, to which reference has been made. When the specimens are assembled according to the stage of general skeletal maturity and not according to chronologic age, the great variation, herein defined as discrepancy, becomes insignificant. It is then possible to establish for each skull a maturation value on the basis of a common time unit, comparable to those manifest in unretarded children in good health. The effect of time itself on any living creature depends in the earlier phases on the relative rate of growth and maturation (development) dictated by conditions of health and environment. In later phases of life, however, it covers merely recurrent injury and repair, which does not necessarily mean the approach of senility, unless some adverse complication disturbs the delicate balance between these two processes. However, in this study there is no need to elaborate on any other than the earlier phases of life. In these phases there is no distinction between white and Negro children, so far as the influence of health, maturation or time is concerned.

PHASES OF DEVELOPMENT BASED ON AMOUNT OF PNEUMATIZATION

There are four phases, as follows:

I. Pneumatization commencing (birth)
II. Pneumatization moderate (2 years)
III. Pneumatization extensive, not including apex (childhood, 6 to 14 years)
IV. Pneumatization complete, including apex (adolescence)

Considering the phases of development based on the extent of pneumatization, I shall begin at the earliest age in my series. Specimen 1885, of 7 fetal months maturation age, shows in the roentgenogram (fig. 4 *A*) the first turn of the cochlea at the apex of the pyramid. There is little bony extension lateral to the semicircular canals, which are clearly

shown. The cavity of the middle ear appears plainly, showing its outline and part of its contents, namely the malleus and the incus. At this stage, there is also noticed a backward extension to the tympanic cavity, representing the mastoid antrum. To summarize, at this stage one finds

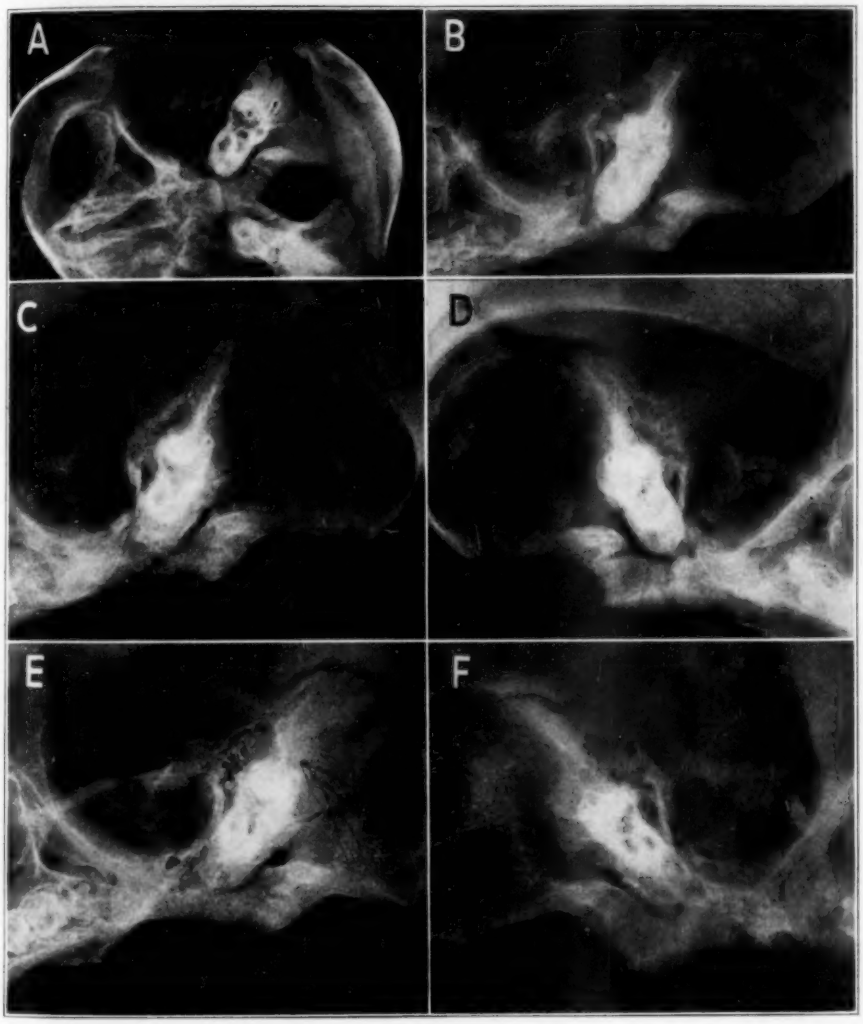


Fig. 4.—*A*, skull 1885, 7 fetal months maturation age. *B*, skull 1462, birth maturation age. *C*, skull 232, 1 month maturation age. *D*, skull 230, 2½ months maturation age. *E*, skull 818, 4 months maturation age. *F*, skull 1148, 6 months maturation age.

fully developed the internal ear and the middle ear with its contents. Other specimens studied at this stage of development are specimens 1845, 2472, 2431, 2271, 2201, 2149, 1620, 1919, 2119 and 1465.

As development progresses from this stage to the birth stage, there appears at the apex of the pyramid a bony extension medial to the first turn of the cochlea. There is increased formation of bone external to the internal ear, but there is no definite bony union between the pyramidal and the squamous portion, and along this suture there already appears a definitely outlined area of pneumatization, hereafter called the area of differentiation, which is connected with the mastoid antrum and the cavity of the middle ear. This stage is exemplified by specimen 1462 (fig. 4 *B*) and is found also in specimens 1689, 2134, 2183, 273, 278 and 240. As an example of the 1 month stage, specimen 232 is typical (fig. 4 *C*). It shows a bony extension medial to the first turn of the cochlea as a developing apex to the pyramid. The external portion of the pyramid extends to meet the parietal and the occipital bone. It becomes more difficult to discern the suture between the squamous and the pyramidal portion of the temporal bone. The area of differentiation becomes more clearly outlined and may even be pneumatic.

Specimen 230 is an example of the 2½ month stage (fig. 4 *D*). This specimen shows a greater length of the apex or carotid portion of the pyramid, and the area of differentiation is definitely pneumatic. Henceforth it will be called the area of pneumatization. The spaces between the temporal and the occipital bone have become smaller, and the squamous and the pyramidal portion of the temporal bone are definitely united at this stage. Specimen 818 (fig. 4 *E*) shows the normal development for the 4 month stage. The tangent of the first turn of the cochlea is at least 8 mm. from the tip of the apex. The peripheral portions of the temporal bone now fill the spaces along the suture between it and the parietal and the occipital bone. The opening for the emissary vein is easily visible. The area of pneumatization also is clearly outlined and extends from the middle ear laterally into both the pyramidal and the squamous portion, but mostly into the squamous portion. It extends along the line of union between the squamous and the pyramidal portion and is completely anterior to the posterior margin of the pyramid. Pneumatization at this stage is entirely external to the middle ear; no evidence of it exists in the apical portion of the pyramid.

The left temporal bone of specimen 1148 (fig. 4 *F*) represents the normal development of a temporal bone at the 6 month stage The area of pneumatization extends posterior to the posterosuperior margin of the pyramid. This posterior extension on the roentgenogram indicates a downward and peripheral extension toward the mastoid process, which at this stage is rudimentary. This area of pneumatization has definite trabeculae, outlining each cell. One must bear in mind the position of the pyramid when considering the direction and extension of pneumatization. The area of pneumatization from one stage to another does not

seem to change much in size on the roentgenogram because such extension as occurs lies directly in the axis of the central ray of the x-ray beam.

If specimen 1950 R (fig. 5 *A*) is taken to show the normal development of the temporal bone at the 13 month stage, the tangent of the first turn of the cochlea is found to be 15 mm., more or less, from the tip of the apex. The wall of the canal of the internal carotid artery can be traced throughout the apical portion of the pyramid. The temporal bone now articulates closely with the parietal and the occipital bone. The area of pneumatization is confined to the external portion of the pyramid and shows many trabeculae radiating away from the zone adjoining the mastoid antrum. The outline of the internal ear remains clear, and the small rudimentary mastoid process is visible but at this stage contains little if any pneumatization.

Specimen 1385 represents the 1 year 7 month stage (fig. 5 *B*). It shows a well developed apex of the pyramid, which appears identical in bony texture with the basilar portion of the occipital bone. The area of pneumatization now can be seen to extend into the mastoid process, into the posterosuperior angle of the pyramid, into the squamous portion and even toward the root of the zygoma. In the area of the mastoid antrum there are many fine bony trabeculae, but as these extend peripherally they separate more and at the edge of the pneumatic area form the outlines of large cells. Specimen 1115 (fig. 5 *C*) illustrates findings at the 3 year 11 month stage. This example differs from specimen 1385 only in the extension of pneumatization into the mastoid process, which cannot be demonstrated in the roentgenogram because it occurs in the axis of the central ray. The sutures between the temporal, the parietal and the occipital bone, it is true, show more bony union. There is the additional observation in specimen 1115 that the pneumatization consists of many small cells, but this is unusual at this stage. The apical portion of the pyramid shows clearly the canal of the internal carotid artery and cancellous bone identical with the basilar portion of the occipital bone.

Specimen 2240 R (fig. 5 *D*) demonstrates that at the skeletal age of 4 years 8 months features are precisely those of 3 years 11 months with the exception that the mastoid process is more developed and the trabeculae are less clearly visualized. The apical portion of the pyramid is identical in texture with the basilar portion of the occipital bone. Next to be considered is the skeletal age of 5 years 8 months, illustrated by specimen 1074 (fig. 5 *E*), which shows almost complete pneumatization of a well developed mastoid process. The tangent of the first turn of the cochlea is now between 15 and 20 mm. from the medial tip of the apex. No further significant increase in this dimension occurs with succeeding

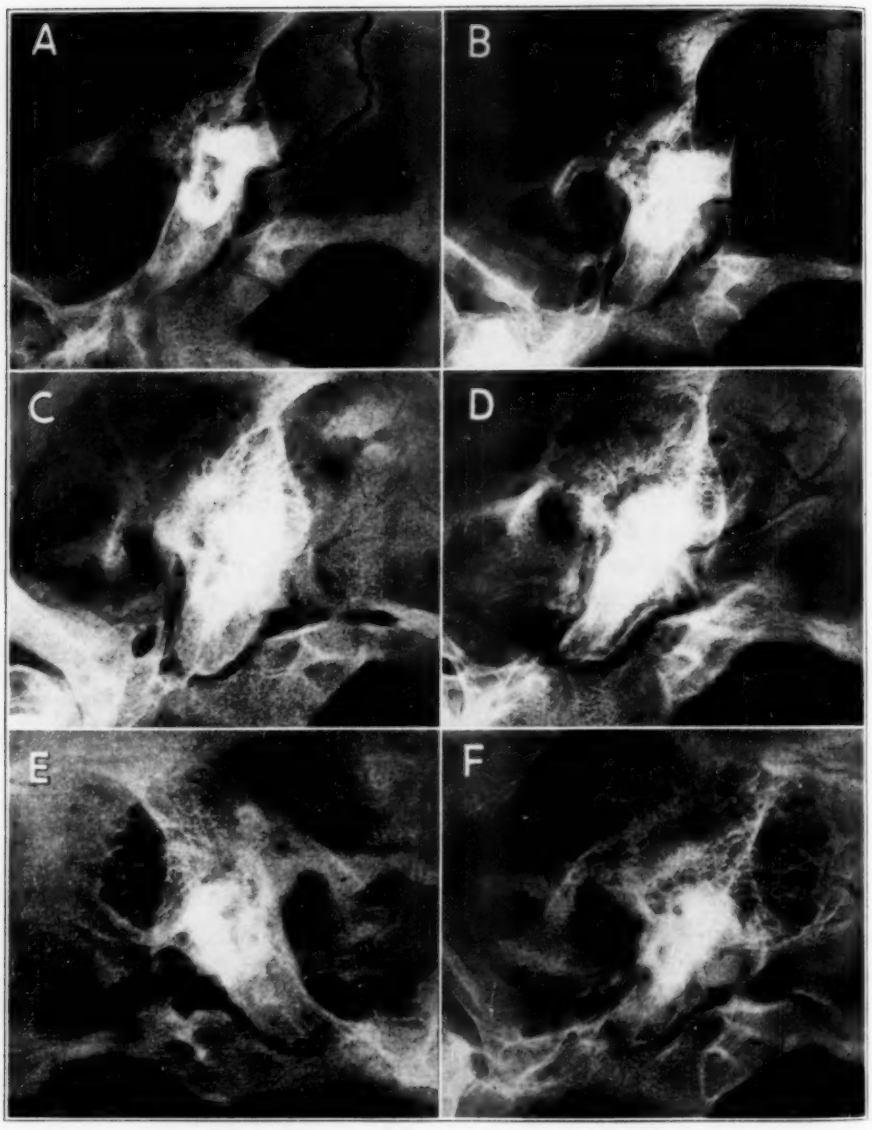


Fig. 5.—*A*, skull 1950, 1 year 1 month maturation age. *B*, skull 1385, 1 year 7 months maturation age. *C*, skull 1115, 3 years 11 months maturation age. *D*, skull 2240, 4 years 8 months maturation age. *E*, skull 1074, 5 years 8 months maturation age. *F*, skull 632, 6 years 9 months maturation age.

years. The canal of the internal carotid artery is clearly outlined, and the rest of the apical part of the pyramid appears identical in texture with that of the basilar portion of the occipital bone.

Specimen 632 (fig. 5*F*), skeletal age 6 years 9 months, shows pneumatization extending throughout the mastoid process, back almost to the suture between the temporal and the occipital bone, forward into the root of the zygoma and up into the squamous portion of the bone. In other words, there is extensive pneumatization of the external portion of the pyramid and its adjacent parts. The apical portion of the pyramid appears identical in texture with the basilar portion of the occipital bone. There is no evidence of pneumatization in this apical portion. Specimen 872 L (fig. 6*A*), of 9 years 8 months skeletal age, shows extensive pneumatization in the lateral or external portion of the pyramid, including the mastoid process and the squamous portion, extending backward toward the suture with the occipital bone and forward into the root of the zygoma. At this stage there is no evidence of pneumatization in the apical portion of the pyramid, which appears identical in texture with the basilar portion of the occipital bone.

Specimen 1240 (fig. 6*B*) has a skeletal age of 13 years 8 months. At this stage there is extensive pneumatization of the external portion of the pyramid but no more than at the previous stage. This specimen shows no evidence of pneumatization of the apical part of the pyramid, which is identical in texture with the basilar portion of the occipital bone. Specimen 1041 (fig. 6*C*) has a skeletal age of 15. It shows no significant changes from specimen 1240. The apical portion of the pyramid still is identical in texture with the basilar portion of the occipital bone. Specimen 527 L (fig. 6*D*) shows pneumatization extending throughout the external portion of the pyramid, with ramifications backward toward the occipital bone and into the mastoid process, upward into the squamous portion and forward into the root of the zygoma. This specimen comes from an epileptic girl of skeletal age 16. It presents pneumatic cells in the apical portion of the pyramid. These cells are clearly outlined by bony walls; they do not extend throughout the entire apex of the pyramid but are present in its posteroinferior portion. Hence the texture of the apex of the pyramid is now distinctly different from that of the basilar portion of the occipital bone.

Specimen 807 (fig. 6*E*) is taken from a boy of skeletal age 17. It shows pneumatization in the external portion extending well anterior into the root of the zygoma, into the squamous portion of the bone and into the mastoid process. The entire apical portion, except that occupied by the internal carotid artery, is completely filled with pneumatic cells. The texture of the pneumatic apical portion, therefore, stands out in

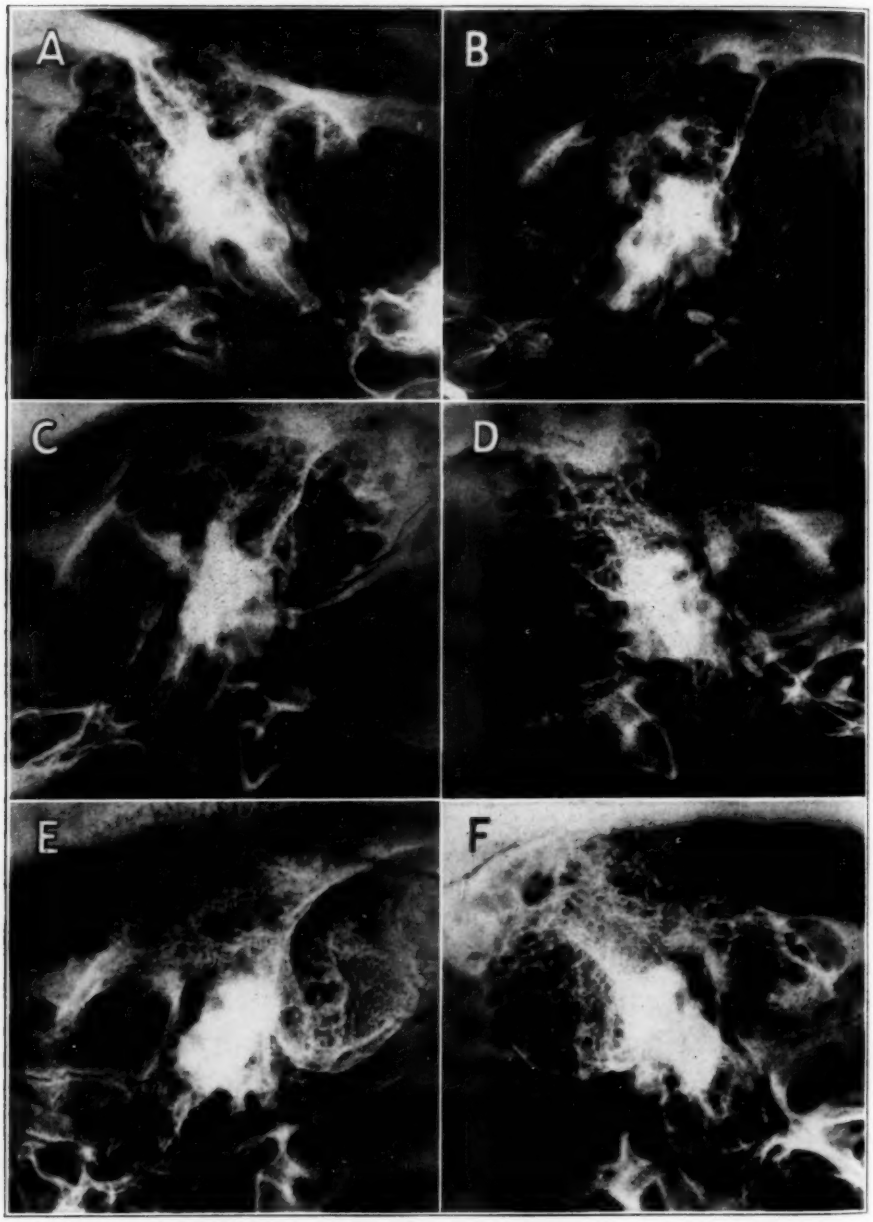


Fig. 6.—*A*, skull 872, 9 years 8 months maturation age. *B*, skull 1240, 13 years 8 months maturation age. *C*, skull 1041, 15 years maturation age. *D*, skull 527, 16 years maturation age. *E*, skull 807, 17 years maturation age. *F*, skull 503, 20 years maturation age.

marked contrast to the cancellous basilar portion of the occipital bone. Specimen 503 (fig. 6*F*), taken from a white man of skeletal age 20, shows a well developed temporal bone with a large mastoid process. Pneumatization completely fills the mastoid process and extends well back toward the suture with the occipital bone and up into the squamous portion nearly to the suture with the parietal bone. A large cell appears in the posterior end of the zygomatic arch; cells appear even in the postglenoid process. The apical portion of the pyramid is completely pneumatized. Therefore, this is one of the best examples this study has produced.

Up to this point the report has been concerned with establishing a standard for the various stages, the maturation ages being taken as arbitrary distinguishing criteria. The earliest specimen that shows pneumatization medial to the internal ear is 527, 16 years of age. It is of course possible that pneumatization may reach the apex in certain persons before this age, though I have not been able to illustrate it in any of the specimens. The material is sufficient for the establishment of a working schedule, arranged according to the amount of pneumatization and divided into the following phases:

I. Birth. In this phase one meets the picture represented in specimen 1462; it has been described previously, but for the sake of completeness it will be mentioned here that in this example pneumatization occurs only in the immediate vicinity of the mastoid antrum.

II. Approximately 2 years. This phase is represented by specimen 1385, which happens to have a maturation rating of 19 months. It shows pneumatization extending into the squamous portion and into the rudimentary mastoid process.

III. Childhood (6 to 14 years). In this phase there is extensive pneumatization except in the apex. It is represented by specimens 632, of 6 years 9 months maturation age, and 1240, of 13 years 8 months maturation age.

IV. Adolescence (16 to 20 years). In this phase the pneumatization extends both externally and medially to the internal ear. Pneumatization of the apex may be extensive or small in amount, but the process for each person seems to be completed by this age.

Thus a standard based on maturation age is developed by a study of specimens selected from those which show the most extensive pneumatization for each stage. Investigation based on these specimens is not handicapped by any evidence of retardation in the process of pneumatization. Such a selection of material is necessary because pathologic causes have hampered the development of the air cells in many otherwise acceptable examples. The next step is the classification of adult mate-

rial according to the scale of standards developed in this section. This material is divided into four groups or phases, roentgenologically, according to the amount of pneumatization. As roentgenology is readily applicable to the living, one can, through its aid, estimate the probable findings within the bone before actual operation. One can, moreover, understand the various clinical pictures mentioned by Wittmaack,[2] who stated that normal development and pneumatization continue throughout life, but when pneumatization is retarded the development is arrested in the phase in which the retardation happens to occur. Hence some specimens from persons of advanced age show pneumatization arrested at birth; others show it arrested before or at the age of 2 years, and still others, in later childhood.

(To Be Concluded)

PSYCHOLOGY OF LARYNGECTOMIZED PATIENTS

LEROY A. SCHALL, M.D.
BOSTON

From the earliest writings to the most recent literature concerning cancer of the larynx, the operation of total laryngectomy is ofttimes described as a mutilating procedure leaving its victim in a pitiable condition—a condition that is worse than death. The psychic reaction to the loss of the power of speech, it is said, frequently leads to suicide or to insanity.

The medical profession and the public are familiar with the blind, with the deaf and with the cripple. Neither the public nor the vast majority of physicians are familiar with the patient who has lost his larynx and consequently normal speech. It is a human failing to dread the unknown. There is need of educating the medical profession as well as the laity to an understanding that the laryngectomized patient is not a pitiable object, a woebegone creature who has given up all that life holds dear. It is hard properly to evaluate human suffering. The blind say they would rather be blind than deaf; the deaf, that they would rather be deaf than blind; the patient without a voice considers himself fortunate that he is neither blind nor deaf.

In advising the total removal of the larynx, one must appreciate the readjustments the patient must make, and one must evaluate the patient's ability to make these adjustments. His mental outlook should be given serious consideration. The patient who lives wholly within himself, the self-centered, brooding person, whose world revolves around himself, the introvert, who faces any crisis with the greatest difficulty, is poor material to make the mental readjustment necessary after total laryngectomy.

I am told that the most frightful and distressing moment is that when the patient comes out of the anesthetic, for then the force of the conditions under which he will have to live for all time is suddenly thrust upon him. The inability to feel air pass through the nose or mouth during respiration is most distressing. Through the inability to inhale through the nose, the sense of smell is greatly impaired, robbing one of much of the enjoyment of food. Naturally with some foods this matters little, but the laryngectomized patient must become reconciled to eating foods whose taste seems changed or whose flavor is greatly

Read in a symposium on laryngeal cancer at the Forty-Fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., April 28, 1938. Papers by Henry Boylan Orton and William Harris and Paul Klemperer were published in the September issue.

diminished. The larynx, then, too, plays an important part in swallowing. Without the larynx, when one eats too rapidly or fails to masticate thoroughly, the food lodges in the throat and can be dislodged only by some liquid.

After the patient has learned to talk either with the "esophageal voice" or with the artificial larynx, he is subjected to curiosity which is not always pleasant, as comparatively few people are familiar with an artificial voice. Then, too, because the patient cannot talk normally, it seems to be a natural reaction to assume that he cannot hear, and he is shouted at unnecessarily.

After the operation the realization of these handicaps gradually grows on the patient, so that by the third or fourth postoperative day, he frequently becomes depressed and despondent, a type of psychotic disturbance that might be termed a reactive depression. All visitors except the immediate family should be excluded, and the patient's despondency must not be reflected by the attending nurses or physicians. Should the patient continue to overemphasize his loss, he would before long find himself in a mental condition from which he might be able to extricate himself only with the greatest difficulty. It is best to accept it as a loss and start anew. It is advisable to explain all this to the patient previous to the operation, so that he can intelligently face these annoying factors. It is especially necessary to explain to the patient before the operation that he will in a short time be able to make himself understood, either by the use of an artificial larynx or by the development of an esophageal voice; and this achievement must be held up as a distinct goal to obtain. What one must do is to offer a positive substitution for the lost speech, rather than the ordinary negative consolation of, let me say, simply saving one's life. As soon as possible, then, the patient should be supplied with an artificial larynx and instructed in its use.

It is said, too, that to make these readjustments the patient must be either very intellectual or very ignorant. This has not been my experience with the laryngectomized patients. If the patient can develop the mental attitude of looking ahead to the years to come, instead of backward to the years before his affliction, if he can develop the attitude that "although I am handicapped, I have conquered," and if he will use his artificial larynx or esophageal voice just as the blind use a seeing eye dog, the deaf a hearing aid and the lame a crutch, in time he will be neither a curiosity to his friends nor objectionable to those with whom he comes in contact. There may be periods of depression, but these will be of no greater frequency than those that come to any one.

Although handicapped, the laryngectomized patient must have the desire to live. He must have interests outside himself. His outlook on life must be forward, not backward. A physician with a fine practice, a

patient whose wife had double cataracts, an optician who loved to hunt, a farmer with his farm and live stock, a master mechanic in charge of a machine shop, a plumber with his trade are but a few examples of patients who had the incentive that prompted them to make the necessary readjustment. The extrovert makes this readjustment most readily and takes great pride in his accomplishment.

There is, of course, no reason why a laryngectomy, like any other operation, might not act as a precipitating factor for a psychotic episode of greater or less gravity. The type of reaction shown by the patient will depend largely on other factors; in other words, there is no specific type of mental disease or mental symptom which one would look for after an operation of this sort.

Despite the emphasis placed on suicide following laryngectomy, it is no more frequent than that following many other diseased conditions. Among my patients, there has been but a single suicide—and this was not on account of the loss of voice but due to an incurable recurrence. This patient was a master mechanic in charge of a machine shop with 40 men under his direction. For two years he carried on efficiently and satisfactorily, and he took his life only after a recurrence developed in the skin of the neck and upper part of the chest and after he had received the maximum of radiotherapy without benefit.

In 102 cases in which laryngectomy was performed Orton [1] has had no patient who committed suicide or suffered mental derangement. After his review of 800 cases of cancer of the larynx, Jackson [2] stated: "The records do not show any case in which mental derangement or suicide occurred." In a later communication Jackson [3] supplemented this statement by saying:

> On going back over the records, I have found 2 cases in which the patient committed suicide. Neither of them had had a laryngectomy. One was under treatment by irradiation. The other committed suicide 11 years after laryngofissure. There was a good voice, and there was no recurrence. In the opinion of the coroner's physician, the suicide was due to financial worries.

Insanity precipitated by total laryngectomy must be rare.

Inquiry of forty-nine hospitals for the insane, with a total bed capacity of 113,926, reveals that in the entire history of these institutions but 2 patients were admitted in whose cases the operation may have been a factor.

REPORT OF CASES

CASE 1.—The patient was in a state hospital for persons with mental disease from June 12, 1920, to May 27, 1922, when he was discharged as recovered from a manic-depressive type of insanity. He was seen by a laryngologist on Sept. 9,

1. Orton, H. B.: Personal communication to the author, March 16, 1938.
2. Jackson, C.: Personal communication to the author, March 10, 1938.
3. Jackson, C.: Personal communication to the author, May 31, 1938.

1926, giving a history of hoarseness of two years' duration. Total laryngectomy was performed on Nov. 11, 1926, and on Dec. 13, 1926, the patient was readmitted to the hospital for the mentally diseased, where he remained until his death, on June 26, 1933. It is believed that the laryngectomy was the precipitating cause of his second attack.

CASE 2.—From New York state, the superintendent of a hospital under the Department of Mental Hygiene wrote: "A patient has recently been admitted who underwent a laryngectomy a few years ago. As yet no detailed history of his life is available, and it is impossible to tell whether the laryngectomy was the cause of his mental condition."

CONCLUSIONS

For the properly selected patient, laryngectomy, when indicated, is not a prominent factor in precipitating the act of suicide or in causing mental derangement in the after-life if no previous psychopathic state has existed. Contact with a fairly large group of laryngectomized patients shows them to be a most optimistic, cheerful group, neither objects of curiosity nor, in most cases, recipients of charity.

INDICATIONS FOR DIFFERENT TYPES OF TREATMENT OF MALIGNANT DISEASE OF THE LARYNX

CHARLES J. IMPERATORI, M.D.
NEW YORK

Evaluation of the indications for any specific measure in the treatment of cancer of the larynx, whether involving surgical intervention or irradiation, may be arrived at by consideration of the following diagnostic postulates. Under the circumstances, generalities are necessary, but each case must be considered individually.

DIAGNOSTIC CONSIDERATIONS

Individual History.—The history of the patient, including his occupational and particularly his laryngeal history, the degree of abuse of the voice, laryngeal lesions, such as papilloma, chronic laryngitis, alcoholism, excessive use of tobacco, dental decay and poor oral hygiene are to be considered from a local standpoint. General diseases, such as tuberculosis and syphilis, should be considered not only for differentiation but as possible accompanying diseases. A family history of parents or near relatives having had cancer, in my experience, should not be considered too seriously as evidence that a given lesion is malignant. However, as the cause of cancer remains unsolved, consideration must be given, as an etiologic factor, to the possible vulnerability of certain families to cancer. To quote from Chevalier Jackson,[1] the etiologic factor may be expressed in an algebraic formula. Age, plus sex, plus senile changes in epithelium, plus irritation, plus frustrated repair, plus alcohol, plus tobacco smoke, plus XYZ, the as yet undetermined factors, equals etiology.

Laryngeal Inspection.—Laryngeal study, particularly by the indirect method, should be such that examination shall visualize the introitus of the larynx in its entirety and especially the anterior commissure. The ventricular bands and vocal cords are examined for limitation of motion. All abnormalities in loss of continuity of the normal structures must be considered. The location of a new growth, its size and

---

Read in a symposium on Laryngeal Cancer at the Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., April 28, 1938.

1. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 378.

appearance and the apparent involvement of the adjacent structure require experience to evaluate. A growth in the anterior commissure, particularly if slightly bilateral, is not as good a risk for any therapeutic measure as one located in the midpart of a cord, while a growth in the posterior commissure may already have metastasized. This condition is dependent on the lymphatic circulation of the laryngeal structure. Palpable lymph nodes in the neck and a malignant lesion in the larynx cannot be looked on without alarm. Direct examination, except for gross lesions and subglottic invasion, is not as satisfactory as one feels that it should be. Loss of mobility of the cord is significant of a lesion fairly well advanced or of a subglottic growth not seen by indirect laryngoscopic examination. A benign lesion, involvement of the cricoarytenoid joint or paresis of the cord may produce loss of motility. No importance should be attached to the absence of loss of mobility in the diagnosis of carcinoma of the larynx.

Biopsy.—Biopsy for histologic grading can best be done by the direct method, with the Jackson anterior commissure speculum and a triangular basket punch type of forceps, such as mine. The biopsy specimen should, if possible, include tissue adjacent to the new growth, except in instances in which there is a definite doubt that the growth is now malignant. In that event, only the redundant tissue should be removed.

Histologic Grading.—Irrespective of the storm of pathologic controversy regarding the attempted grading of malignant growths, it is felt that the grading of these growths by Broder's method has a great deal of merit and has been of great assistance to the laryngologist. Definitely, growths roughly graded and classified as 1 or 2 are the type of malignant growths that are amenable to surgical intervention or irradiation, with a reasonable amount of assurance to the patient that there will be no recurrence within five years. The grading of a growth as 3 may apply only to a local area included in the material taken for biopsy. Growths graded as 4 respond rapidly to irradiation but as a rule have already metastasized. It is recognized that frequently in the same growth there will be variation in the cells and therefore a variation in the classification.

Fluoroscopic Examination of the Larynx.—Fluoroscopic examination of the larynx and adjacent tissue should be done as a routine to determine the departure from normal of the mobility of the laryngeal structures during respiration, phonation and deglutition.

Roentgenographic Examination.—The roentgenogram should substantiate a further study of the epiglottis, the ventricle and the laryngeal encroachment in the laryngopharynx and the subglottic space.

Clinical Course of the Disease.—The clinical course of the disease should be noted, including the duration of hoarseness, if any, dyspnea and dysphagia and response to the previous manipulation, whether it consisted of surgical or local treatment.

INDICATIONS FOR SURGICAL TREATMENT

Surgical treatment consists of one of two procedures, thyrotomy and laryngectomy, and should be applied to intrinsic growths. Extrinsic growths are those found on the aryepiglottic folds, the epiglottis, the adjacent mucosa of the laryngopharynx or the posterior cricoid area. They usually are extensions of growths from within the larynx, the exception being postcricoid growths. All these types of growths, as a rule, are accompanied by adenopathy. So-called inoperable growths consist of those growths that have extended from the larynx into the adjacent tissue and are extensive in area. Marked difficulty in swallowing and laryngeal obstruction characterize these growths. Surgical treatment for extrinsic growth is occasionally crowned with success, but usually the results obtained are but palliative.

Endolaryngeal operations, other than removal of tissue for biopsy, should not be done. Electrosurgical procedures may be combined with the use of the scalpel.

Thyrotomy or Laryngofissure.—The indications are as follows:

1. A unilateral cordal growth located anterior to the vocal process and involving not much, if any, of the anterior commissure.

2. Histologic grading of 1 or 2 by Broder's classification; squamous cell carcinoma in an adult.

3. Slight or no loss of mobility of the cord.

4. Previous clinical course and individual history as noted in the introductory section.

5. No adenopathy.

Laryngectomy.—The indications are as follows:

1. An intrinsic lesion of the larynx of such size, location and involvement of the structures that the operation of thyrotomy would be insufficient; no palpable lymph nodes.

2. A bilateral anteriorly located lesion in certain cases. This is a borderline condition, and the cases must be dealt with individually. A thyrotomy may result in a cure. All posterior lesions demand total extirpation.

3. Unsuccessful irradiation accompanied by perichondritis and necrosis of the framework of the larynx. Life may be prolonged for

a period of from one to two years by laryngectomy, but it is rarely curative.

4. Extrinsic lesion of the larynx. This condition, unfortunately, rarely yields to any known method of treatment.

INDICATIONS FOR IRRADIATION

The proper evaluation of irradiation has not been given prominence because of failures, mainly due to improper selection of cases and to other factors, such as improper apparatus and dosage. The method of choice is that of Coutard. It would seem that if cases were selected sufficiently early in the disease results might be obtained that would compare favorably with those obtained by surgical intervention. Irradiation is not without extreme discomfort and certainly when used by the fractional method of Coutard is not without consumption of time. Interruption of the course of irradiation because of tracheotomy may destroy the result of the treatment and entail the loss of valuable time. Careful consideration should be given to the degree of stenosis before one proceeds with irradiation. Indications for this method are:

1. A basal cell growth.
2. An epithelioma on a noninfiltrated cord.
3. A growth in the epiglottis or the glossoepiglottic folds.
4. An inoperable extrinsic growth evidenced by enlarged lymph nodes; postcricoid growth.

Irradiation may be used also after thyrotomy or laryngectomy.

COMMENT

Cancer of the larynx is a serious disease, for it is rarely discovered early, regardless of all the "shouting from the housetops" of laryngologists to their medical colleagues who first come in contact with the patient. Chevalier Jackson's postulate that it is one's duty to prove every laryngeal growth either malignant or nonmalignant apparently remains unheeded, and applications of silver nitrate or instillations of oils are substituted for a proper investigation, so that precious time is lost before the correct diagnosis is reached. Whatever treatment is applied in the individual case must be early in order to secure a measurable degree of success.

MENINGITIS FROM THE SPHENOID SINUS

R. WALLACE TEED, M.D.
ANN ARBOR, MICH.

From the time of its description by Fallopius, in 1561, to the publication of Max Schaeffer's paper on diagnosis and treatment, in 1890, interest in the sphenoid sinus was largely centered on the anatomy and pathology. The dictum of Hyrtl that *"Die Keilbeinhöhle ist ganz ausser dem bereiche chirurgische Angriffe"* (The sphenoid sinus is entirely outside the domain of surgical attack) was universally accepted.

Although Cavanaugh and others said they believed that the first known case of disease of the sphenoid sinus was that described by Rouge, in 1871, investigation has revealed several earlier reports. The earliest one of which I am aware was that of Lieutaud, in 1735. The patient was supposed to have had pulmonary suppuration, but autopsy revealed a purulent disorder of the maxillary, frontal and sphenoid sinuses.

A report published in 1810 by Leveque-Lasource concerned a woman 38 years of age. She complained of headache, anorexia and vertigo. She became blind, first in the left and then in the right eye. Deafness in the left ear next appeared, followed by paralysis on the left side, at first partial but later complete. Deafness then appeared in the right ear, and the voice became altered. She gradually weakened and died about fifteen months after the onset of the symptoms. Postmortem examination revealed that the pituitary gland was twice the normal size and contained several fetid abscesses. The sella turcica and the posterior clinoid processes were almost completely destroyed. The sphenoid sinuses were filled with pus, and they communicated with the cranial cavity. In the left middle fossa was a tumor which originated in the internal acoustic meatus and occupied three fourths of the posterior face of the petrosa. The tumor filled the orifice of the meatus and destroyed its walls. While it is not so stated, this tumor was undoubtedly part of an extradural abscess originating in the sphenoid sinus.

Abercrombie, in his book published in 1843, described a case noted in 1820 which probably represented a case of infection of the sphenoid sinus with extension to the orbit and the meninges. The patient, a woman aged 22, complained of the sudden onset of pain in the left temple, with some extension to the vertex. The following week swelling appeared in the left upper lid, accompanied by chills. Puncture of the swelling released pus, and a probe revealed denuded bone along

the orbit. Two days later she had a slight convulsion. Afterward she complained of pain in the crown of the head. Three days later she died. Autopsy revealed an extradural abscess at the base of the brain; the right hemisphere was covered with thick pus. There was an orbital abscess, but no disease of the bones of the orbit was found. The sinuses were apparently not opened.

Palletta, in 1820, observed a child aged 10 with a polyp in the left side of the nose. Partial extirpation was done, but the polyp recurred and became voluminous, obstructing respiration, deglutition and speech. It was again removed; this time otorrhea occurred, followed by exophthalmos on the right and death. Autopsy revealed pus at the base of the brain and gangrene of the meninges, the middle lobe of the brain and the pituitary gland. Extending from the mucosa of the sphenoid sinus and greatly enlarging its orifice was a large polyp.

In spite of these cases, Mackenzie wrote in 1833 that he knew "of no instance on record in which the sphenoid sinuses were dilated by inflammation or polypus." He said he believed, however, that "they could expand easily . . . either upwards or outwards . . . [and] deform the posterior part of the orbit."

Andral, writing in 1834, described a case observed in 1820. The patient, a man of 64, was brought to the hospital in delirium after an illness of fourteen days. Two days later, spasm of the muscles on the right side developed with pleurothotonus, and he died. Autopsy revealed creamy pus over the lateral and superior surfaces of the hemispheres. The pituitary body was entirely destroyed by pus.

The case observed by Forget, in 1850, was similar to that of Palletta. A boy aged 15 had a polyp of ten years' duration which caused deformity of the nose and face and projected out of the right side of the nose. There was exophthalmos on the right and depression of the palate. An attempt was made to remove the tumor through an external approach, but death occurred five days later. Autopsy revealed destruction of the turbinates, the vomer and the perpendicular plate of the ethmoid. There was perforation of the cribriform bone on the left, although the dura covering it was intact. A large polyp was attached to the basilar apophysis and the great wing of the sphenoid. The sphenoid sinuses were filled with morbid tissue which raised the sella so that its excavation was effaced. The second, third and fifth nerves were pressed against the base of the brain. In the right middle fossa was a mamillated tumor arising from a perforation in the great wing.

Popham, in 1855, reported the case of a girl aged 10 who suddenly became blind; two weeks later convulsions, coma and paralysis developed. Death occurred in thirty-six hours. At autopsy the arachnoid was inflamed, and a cheesy purulent tumor, apparently growing out

of the pituitary body, was seen compressing the chiasm. The sphenoid sinus contained pus, and the body of the bone was carious, the roof being cribriform. No evidence of tuberculosis could be found in the lungs or elsewhere.

The case reported by Simon, in 1858, is interesting in that it is probably the first one known in which disease of the sphenoid sinus was complicated by multiple abscess of the brain. A man aged 50 had had a polyp removed from the nose on several occasions. He began to be deaf and soon lost vision in the right eye. Later, blindness developed also on the left. Repeated epistaxis was followed by epilepsy, left hemiplegia and death. Autopsy revealed basal meningitis. In the right hemisphere were three collections of pus, one close to the upper surface, another about the middle of the external surface and a third deeply situated near the corpus striatum. The second was the largest, being about the size of a hen's egg. Each abscess was bounded by soft brain tissue. The sella, basilar process and posterior clinoid processes were necrotic and sequestrated. The sphenoid sinus was occupied by a polypus which extended into the nose. The cribriform plate was brittle, and the dura over it was healthy. The right cavernous sinus was thrombosed, as well as the internal carotid artery within it. The bone of the internal wall of the cavernous sinus was completely absorbed.

In 1863 Horner reported the case of a girl of 15 who complained of headache for some time and finally became blind. Exophthalmos appeared on the right, and she died. Postmortem examination showed meningitis, but the brain substance was normal. The whole basilar process of the sphenoid, both wings and the roof were carious. There was marked destruction around the right optic foramen, with thickening of the optic nerve.

Demarquay, in 1869, saw a man aged 74 who, four years previously, had an abscess of the frontal sinuses. Trepanation was followed by healing. At 73 a polyp in the nose was removed, followed by erysipelas of the face. Six months later the polyp recurred and removal was again followed by erysipelas. Three months later recidivation occurred, with deafness, epistaxis and complete nasal obstruction. The polyp was again removed, and three days later the base was cauterized with silver nitrate. That evening severe pain occurred and a week later paralysis of the right eye and lid. Death soon followed. Autopsy revealed that the brain was normal. There was pus in the region of the sella, coming from small openings in the sphenoid. A stylet passed easily through the body of the latter, which was soft, friable and carious. The cavernous sinus was surrounded with pus. The frontal and ethmoid sinuses were also purulent, with pus bathing the lamina cribrosa.

Maas, also in 1869, described the case of a man of 45 who had had six operations for the removal of nasal polypi in ten years. After

the last removal, the polypi recurred, causing obstruction to breathing, epistaxis and vertigo. The polypi not only filled the nose but hung down into the pharynx as far as the epiglottis. Five days after removal of the polypi he became soporific; bilateral exophthalmos developed, and death occurred the next day. Autopsy revealed basilar meningitis and an abscess in the left frontal lobe the size of a pigeon's egg. The lamina cribrosa of the ethmoid and the body and great wings of the sphenoid were almost lacking, and pus had broken into both orbits.

The patient studied by Rouge presented exophthalmos which was thought to be due to maxillary tumor. Operation revealed no particular pathologic condition in the antrum, but the patient died seven days later. At autopsy the sphenoid sinus was seen to be filled with caseous pus, and there was suppuration in the internal and the middle ear. Meningitis was not present.

While acute and chronic infections of the sinuses, in general, are among the most common diseases, infection of the sphenoid sinus is relatively infrequent. Martin studied 31 patients and found that while 15 had sinusitis, the sphenoid sinus was involved in only 1 case (6.6 per cent). He further analyzed 12,000 cases from Lichtwitz' clinic and found 243 cases of sinusitis (2 per cent), the sphenoid sinus being involved in 62 (26 per cent). Wertheim made a study of 360 clinical cases and found 95 cases of empyema of the sinuses (26 per cent), in 35 of which the sphenoid sinus was involved (37 per cent). Yerger (1923) found disease of the sphenoid sinus in 11 of 393 cases of sinusitis (2.8 per cent), and Oppikofer found it in only 3 of 165 cases (1.8 per cent). Lichtwitz studied 149 cases of empyema of the various sinuses, noting involvement of the sphenoid sinus in 30 (20 per cent).

The report of Holmes (1903) is hardly in accord with these reports or with my own experience. He reported 237 clinical cases of sinusitis, in 182 of which (77 per cent) there was disease of the sphenoid sinus. In all the 61 cases of disease of the ear which he reported, the sphenoid sinus was involved. Combining all these reports, with the exception of that of Holmes, it is found that of 1,060 clinical cases of sinusitis, the sphenoid sinus was involved in an average of 15.7 per cent.

When autopsy reports are considered, the percentage of cases of involvement of the sphenoid sinus increases somewhat. Harke, in a study of 400 cases at autopsy, noted sinusitis in 116 (29 per cent). There was sphenoiditis in 31 (27 per cent) of these. Fraenkel examined 63 patients, and found that 40 per cent had some disease of the sinuses, the disease in 17 (71 per cent) being located in the sphenoid sinus. Kicer noted empyema of the sinuses in 88 of 195 patients at autopsy (45 per cent), in 29 of whom (33 per cent) empyema of the sphenoid sinus was present. Kirkland made a postmortem study of

100 cases and observed pus in the sphenoid sinus in 19 (19 per cent). Westenhoeffer (1905) made a postmortem study of 29 cases of epidemic meningitis and observed pus in the sphenoid sinus in 10 (35 per cent). Young studied 30 cases and noted that in 56 per cent there was pus in one or both sphenoid sinuses. Fraser (1924) studied 50 cases, observing abnormal contents in the sphenoid sinuses in 27 (54 per cent). Microscopically, however, inflammatory changes were observed in only 3. Pickworth (1932) studied 317 cases and found one or both sphenoid sinuses infected in 100 (32 per cent); in 4 cases there was no involvement of the sphenoid sinuses.

By adding these figures it is found that in 1,184 autopsies, disease of the sphenoid sinus was noted in 250, or 22 per cent. In the three reports in which the general involvement of the sinuses was mentioned, the sphenoid sinus was reported as involved in 77 of 228 cases (33.8 per cent).

Coming now to a discussion of the intracranial complications of sinus disease, one finds that these are relatively rare in comparison with those of otitits. Gowers collected reports of 240 cases of abscess of the brain, of which only 6 were due to chronic nasal disease. Pitt studied 9,000 autopsy protocols and found 57 cases of intracranial disease of otitic origin, but none originating in the nose. Treitel, in a study of 6,000 autopsy reports, found 21 cases of abscess of the brain, 7 of which were otogenous and only 3 rhinogenous. Wertheim reviewed 10,394 autopsy reports, finding 127 cases of intracranial suppuration, only 6 of which were rhinogenous. In 13,400 autopsy reports Onodi (1908) found 4 cases of rhinogenous abscess of the brain and 45 cases of otogenous complications involving the brain. From 1907 to 1921 Turner and Reynolds (1926 [*a*]) saw 866 patients with suppurative disease of the sinuses. Spontaneous intracranial complications occurred in 6 (0.6 per cent). From the department of pathology of the Royal Infirmary, Edinburgh, they obtained information regarding 3 other cases of rhinogenous meningitis, bringing the total to 9 cases (1.3 per cent). During the same period they saw 8,864 patients with otorrhea, with 201 intracranial complications (2.2 per cent). Maxwell reviewed 1,268 cases of acute and chronic suppuration of the sinuses and found intracranial complications and infections of the blood in 1.7 per cent. In fifteen years at the University of Groningen, Huizinga noted 28 cases of abscess of the brain, all of otitic origin.

Among a total of 28,400 cases reported at autopsy in three of the foregoing reports in which the incidence of rhinogenous and of otogenous intracranial complications was compared, there were 7 of the former to 109 of the latter. In other words, infection extends into the cranial cavity from the ear fifteen times as often as from the nose.

If the incidence of rhinogenous intracranial complications in general is compared with that of intracranial complications originating in the sphenoid sinus in particular, the contrast is striking. In Fraenkel's series there were 5 cases of meningitis, in 3 of which the infection arose from the sphenoid sinus (60 per cent), as it did in 4 of the 6 cases (66 per cent) in Wertheim's series. In 1921 Yerger found that of 290,000 cases from the Cook County Hospital in Chicago, there were 390 cases of disease of the sinuses, the sphenoid sinus being involved in 9 (2 per cent). In 16 of the 390 cases there was intracranial involvement, in 5 originating in the sphenoid sinus (31 per cent). Two years later, Yerger reported 11 cases of disease of the sphenoid sinus, in 7 of which (63 per cent) intracranial complications developed. In 5 of the cases (24 per cent) in Maxwell's series the origin was in the sphenoid sinus. In collected statistics on orbititis following sinusitis by Birch-Hirschfeld, the sphenoid sinus was involved in only 25 of 409 cases (6.1 per cent). Death occurred in 52 cases of this series; 28 per cent of the cases, however, were due to disease of the sphenoid sinus. Key-Åberg stated, on the other hand, that of 20 rhinogenous intracranial complications noted in the Swedish literature, none originated in the sphenoid sinus. Likewise, in Treitel's series, none of the abscesses of the brain was due to sphenoiditis. Combining the foregoing statistics, one reaches the conclusion that, while the sphenoid sinus was involved in about 15 per cent of the clinical cases of sinusitis and in 33 per cent of the cases studied post mortem, it was responsible for approximately 35 per cent of rhinogenous intracranial complications.

The manner in which the complications occur has been a matter for considerable study. Eagleton (1932 [*b*]) stated that infection may reach the meninges (1) from dehiscence, (2) by disease of the bone or (3) by way of a blood vessel.

Dehiscences in various parts of the sinus have been described by numerous authors. Berger and Tyrman noted that the wall between the sphenoid sinus and the optic canal may be thin as paper, and Zuckerkandl, in addition, saw small dehiscences here in some instances. He also described dehiscences leading into the middle cranial fossa, which were covered only by dura. Holmes (1896) found in 50 cases two perforations of the optic canal; Gallemaerts, two in 200 cases, and Onodi (1905), two in 300 cases. Onodi also called attention to small perforations of the wall by blood vessels in this region. Treitel found in 12 cases of sphenoiditis, 4 cases in which perforations of the bony wall were present. Smith (1934) noted, in the course of five hundred operations on the sphenoid sinus, dehiscences of various sizes in 17 cases.

Perusal of the literature does not, however, indicate that developmental dehiscences are important as a cause of intracranial complica-

tions. Two cases (Guibert and Emerson) have been reported in which dehiscences allowed the formation of large aneurysms from which fatal hemorrhage occurred, but practically all the dehiscences mentioned previously were noted solely as anatomic anomalies. The fact that the dura of the middle fossa is exposed is of no great moment, since such a condition occurs repeatedly in mastoid infection and after operation on the mastoid without intracranial extension. The efficient protective mechanism of the dura and other cerebral envelops is well known.

The bony wall of the sphenoid sinus, moreover, does not afford any great protection, since, as Turner and Reynolds (1928 [*a*]) and Eagleton (1932 [*a*]) have shown, perforating vessels pass directly from the mucosa of the sphenoid sinus through the bone to the dura. A similar anatomic condition has also been demonstrated in the frontal sinuses by Sieur and Rouvillois and by Furstenberg. These perforating vessels communicate also with the medullary spaces of the bone and with the arachnoid, whence the infection reaches the bulbar cistern. Pickworth (1932) demonstrated that in the presence of sinusitis, colloidal dyes injected into the subarachnoid space appeared in the olfactory region, in the lateral wall of the ethmoid sinus and in the sphenoid sinus. The dyes frequently passed through thick bone to reach the sinuses. This diffusion did not occur in normal bone, indicating, as Eagleton (1932 [*a*]) pointed out, that infection is often the cause of new communications with the venous plexuses covering the spheno-occipital bone.

The presence of micro-organisms in the mucoperiosteum of the sphenoid sinus and in the bone was first noted, in 1890, by Ortmann. Schlagenhaufer confirmed this observation in 1899. Hajek, in a case of meningitis from the ethmoid sinus, noted cocci not only in the marrow spaces and blood vessels of the bone but also in the vessels of the orbital fat. Pickworth (1929) found organisms, in addition, in the optic nerve, the pituitary gland and the wall of the carotid artery. Some of these organisms exhibited triangular, clubbed or granular forms. Pickworth made Gram stains of thin sections of the sphenoid bone in 200 cases and found gram-positive granules in 80 (40 per cent).

The theory of hematogenous diffusion in infection of the sphenoid sinus is given some support by the work of McMahon on mastoid infection. He showed that infection travels along the submucosa of the tube to the middle ear and mastoid and from thence to the periosteum and marrow. Hadjopoulos and Bell made cultures of biopsy material from the mastoid and found streptococci strictly localized in vascular and lymphatic channels, not diffusely disseminated throughout the tissues. They showed, in addition, that nonmotile organisms do not penetrate tissue beyond exposed surfaces, even though they do possess a fibrinolytic ferment.

Moreover, Eagleton and Peters demonstrated meningococci in the urine of patients who did not have meningitis, indicating a hematogenous invasion. That the so-called gastrointestinal toxemia of infants may be due to disease of the sphenoid sinus as well as that of the mastoid was shown by Pickworth (1935), who found pus in the sphenoid sinus as the only pathologic sign in a child 5 months of age who died with this complex. In another case Pickworth (1932) found similar cocci in the sphenoid sinus and in the wall of the stomach, which also showed hemorrhages.

Thus Eagleton's (1932 [*b*]) three etiologic factors seem in reality to have but one common basis, namely, vascular dissemination. He pointed out that the proximity of the vascular substance of the bone to infection of the vessels of the mucosa makes the base of the sphenoid sinus liable to infective lesions throughout life. In many of the reported cases of intracranial extension from the sphenoid sinus the bone was macroscopically normal. However, histologic examinations have shown microscopic foci of osteomyelitis in such cases, indicating that the time element is of some importance. Jacobsgaard suggested lymphogenous dissemination, but lymphatic vessels in this region have not been definitely demonstrated.

Of the various intracranial complications of disease of the sphenoid sinus, meningitis is by far the most common cause of death. In 1896 Dreyfuss found reports of only 13 such cases in the literature, while in 1906 St. Clair Thomson collected reports of 30 cases, exclusive of cases of syphilitic, tuberculous, malignant and meningococcic involvement. Hajek, in 1915, found reports of 47 cases, and Burger, in 1926, listed only 43. I have been able to locate reports of 129 cases in which the usual pyogenic organisms, i. e., streptococci, staphylococci, Bacillus influenzae, pneumococci, etc., were present. In 52 of these, macroscopic disease of the sphenoid bone was noted, and in 9 cases in which histologic examination was made microscopic evidences of osteomyelitis were seen. Turner and Reynolds (1928 [*a*]) noted erosion of the bone by osteoclasts with the formation of bays. Eagleton (1932 [*b*]) stated that diseased mucosa enters these bays and thus comes into relation with the vascular spaces of the bone. New perforating veins are formed, and these carry the infection farther, into the dura and arachnoid.

In 1 case [1] extension to the meninges occurred through a perforation in the roof of an accessory sphenoid sinus. Grünwald (1902) reported a case in which the normal sphenoid sinus was free from infection, but a tract led from a diseased accessory sphenoid sinus to the vertebral column. The periosteum of the atlas had been destroyed, and there was a fistula into its body. The tooth of the axis was also bare of perios-

1. Druss (case 2).

teum, and the cartilage of the atlantoaxial articulation was partially destroyed. The first case reported by Druss was unique; both sphenoid sinuses were absent, but in the midline of the sphenoid bone there was a tract, about 15 cm. long, leading from the roof of the nasopharynx and containing pus and detritus. The sella was eroded, and the dura

Date	Name
1820	Palletta
1834	Andral
1858	Simon
1863	Horner
1869	Demarquay
1869	Maas
1872	Scholz
1873	Panas
1874	Duplay
1878	Russell
1880	**Leber (case 5)**
1881	Weichselbaum
1883	Weichselbaum
1884	Lloyd
1885	Raymond
1887	**Coupland**
1887	**Netter**
1889	Netter
1890	Ortmann
1892	Thiroloix and du Pasquier
1893	Lyonnet and Regaud
1893	**Pekosławsky**
1893	Zörkendörfer
1894	**Broeckaert**
1895	Dmochowski
1895	Flatau
1895(*a*)	Schwabach
1896	**Fraenkel (3 cases)**
1897	Stoerk (3 cases)
1899	**Caubet and Druault**
1899	Schlagenhaufer
1900	Bertemes
1900	Grünwald
1900	Lodge
1900	Toubert (3 cases)
1900	Vossius
1901	Dodd
1901	Jassniger
1901	Wertheim (4 cases)
1902	Halle
1902	Kander (2 cases)
1902	Kirkland
1902	Wilson
1903	Beek (2 cases)
1903	Jessop
1905	Barnes
1905	Bergengrün
1905	Walb
1906	Thomson
1907	Killian
1907	Schröder
1908	Trautmann
1909	Roughton
1910	Meurers
1911	Gerber
1912	Schulze
1913	Lubliner
1913	Syme
1914	Hilfrich
1915	Imperatori
1916	Knutson
1918	Leegaard (2 cases)
1918	Meyer
1919	Boggs and Winternitz
1919	Vail
1920	Chisholm and Watkins
1920	Leuch
1920	Ruttin
1921	Andrews
1921	Bondy
1921	Fraser
1921	Galtung
1921	Höston (case 1)
1921	Hubby
1921	Key-Åberg
1921	Neumann
1921	Ramdohr (case 1)
1921	Rodger
1921	Yerger
1922	Fremel (3 cases)
1922	Powell
1923	Yerger (4 cases)
1926	Burger
1926	Eagleton
1926(*a*)	Turner and Reynolds
1927	Turner and Reynolds
1927	Vlasto
1928	Bombelli
1928	Turner and Reynolds (2 cases)
1929	Druss (2 cases)
1929	Kramer (4 cases)
1929	Maxwell (5 cases)
1931	Grant
1932(*b*)	Eagleton
1932	Graves
1933	Hesse (4 cases)
1934	Kaufman and Hartmere
1936	Field and Rogers
1936	Smith (2 cases)

over the sphenoid bone was thickened, with meningitic involvement. This tract undoubtedly represented the pharyngeal process described by Rathke, which goes into the formation of the pituitary gland. For some

reason it remained open, became infected and caused meningitis through the development of osteomyelitis.

Extradural abscesses were found in 7 cases [2] in this group. In addition, the literature contains reports of 5 other cases [3] in which no meningitis was found but in which the extradural abscess was believed to be the cause of death.

Cerebral abscess was noted in 7 cases [4] in this series and in 1 other case (Germann) in which the meningitis was serous. Wilson noted a case in which a cerebellar abscess occurred. In addition, 9 cases [5] are recorded in which meningitis did not occur. The location of these abscesses is of some interest. Thirteen were located in the frontal lobe [6] and two in the temporosphenoid lobe (Cange and Yerger), and in 2 cases (Häggström and Andrews) the location was not specified. Schaeffer (1921) noted the intimate relation of the great wing of the sphenoid and the temporal lobe and stated that if pneumatization were expanded laterally into the wing, abscess formation in the temporal lobe might arise. Douglas studied two hundred skulls and found that in only 15 per cent did the sphenoid sinus fail to reach the small wing. However, these anatomic relations have little influence on the location of the abscess, since, as already noted, the majority have occurred in the frontal lobe. This further strengthens the position, as previously stated, that dissemination of infection is predominantly vascular. Extradural abscesses around the alae of the sphenoid, however, do occur.

Suppurative encephalitis occurred in 6 cases [7] of this series and in 1 other [8] in which meningitis was not present. Hemorrhagic encephalitis was noted in 1 case in which there was meningitis (Schwabach, 1895 [*a*]) and in 1 case in which there was no meningitis (Pickworth, 1929), apparently as part of general septicemia.

In addition to the 129 cases of suppurative meningitis, 2 cases (Greene and Germann) of serous meningitis have been reported. Undoubtedly in these cases the meningitis would have been purulent had the patients lived longer.

---

2. Jassniger, Kander, Halle, Schröder, Roughton, Neumann, and Turner and Reynolds (1926 [*a*]).

3. Leveque-Lasource, Forget, Sandford, Vince and Robertson.

4. Simon, Maas, Dmochowski, Wertheim, Leuch, Andrews and Druss.

5. Lebert, Halász, Häggström, McBean, Möller, Cange, Yerger (1923), Lischkoff and Kaplan.

6. Lebert, Simon, Maas, Dmochowski, Germann, Wertheim, Halász, McBean, Möller, Leuch, Druss, Lischkoff and Kaplan.

7. Palletta, Maas, Bombelli, Turner and Reynolds (1928 [*b*]) and Kramer (2 cases).

8. Grünwald, 1900, p. 147.

The association of sphenoiditis and disease of the ear is rather frequent and causes great difficulty in both diagnosis and management. Eagleton (1932 [*a*]) stated that in 29 of 62 cases of meningitis thought to be due to aural disease there was disease of the sphenoid sinus. In the present series of cases, otorrhea was noted in 20 cases, in 17 of which operation on the mastoid was carried out in hope of eradicating the focus, which was later found to be in the sphenoid sinus. Cullom stated that 85 per cent of the cases of infection of the ear result from purulent sinusitis on the same side as the infected ear. Eagleton (1932 [*b*]) said he believed that certain strains of pneumococci and streptococci developing in the sphenoid sinus have a selectivity for vessels which run in pneumatized bone, i. e., mastoid, when discharged into the circulation. The result is that frequently the primary disease of the sphenoid sinus is overlooked. In a few cases in this series it appeared that the disease of the ear and the mastoid was secondary to thrombosis of the lateral sinus, which was caused by an extension of cavernous sinus thrombosis through the petrosal sinus. A number of authors [9] have commented on this problem and have advised a thorough investigation of the sinuses in every case of infection of the ear and mastoid.

In addition to the 129 cases of meningitis already mentioned, the literature contains reports of a number of cases in which specific disease of the sphenoid sinus produced death through this cause. The largest number of cases [10] in this group were those due to the meningococcus. In the next largest group [11] were those cases due to malignant growths of various kinds. Syphilis [12] accounted for 3 deaths and tuberculosis for 1 (Schaeffer, 1892). Actinomycosis was found in 2 cases [13] and streptotricosis in another (Beevor and Buzzard). In 2 cases [14] meningitis was induced by a bullet in the sphenoid.

The relation of the sphenoid to the cavernous sinus is intimate and important. Eagleton (1927) pointed out that the sphenoid sinus is the only sinus in anatomic proximity to a large vein, from which spread of infection is prevented by only a thin layer of compact bone, which is especially liable to death as a consequence of nutritional disturbances. Furthermore, as was demonstrated in 1901 by Sieur and Jacob, numerous anastomosing veins pass from the mucosa through the bone to the

9. Schwabach (1895 [*b*]), Koebel, Fremel, Eagleton (1935), Kramer and Som (1936) and Schillinger.

10. Westenhoeffer (1905, 1906), Embleton and Peters (1915), Embleton (1920) and Peters (1920).

11. Reinhardt, Braun, Macewen, Lodge (case 1, 1902), Avellis (1905), Shambaugh, White, Davison and Kahr, Beck and Guttmann, and Handke.

12. Foucher, Thost and Lodge (1902, case 3).

13. Stevenson and Adair-Dighton, and Kramer and Som (1935).

14. Kander (1902, case 3) and Beek (case 3).

cavernous sinus, so that this portion of the sinus wall is literally a cribriform plate. These authors also demonstrated communications with the orbit and the sella. According to Campbell, all these veins are valveless, so that blood can pass in either direction.

Turner and Reynolds (1927) have maintained that infection around a small vessel produces toxins which pass by transfusion through the wall and injure the endothelium. The latter becomes swollen, and a clot forms. This may become infected and may extend into the cavernous sinus, or bacteria may gain access to the lumen. In 47 of the present series of cases of meningitis there was also purulent thrombosis of the cavernous sinuses. In many instances, other sinuses, such as the coronary, petrosal, sigmoid and lateral sinuses, were also involved. In a number of instances the antemortem symptoms indicated the existence of cavernous sinus thrombosis, but the autopsy did not include examination of this region, so that these cases cannot be included here. In 2 cases reported by Simon and Eagleton (1926) the internal carotid artery was also thrombosed within the diseased cavernous sinus. In 4 cases [15] the longitudinal sinus was also thrombosed. In 11 additional cases [16] there was no meningitis, and cavernous sinus thrombosis constituted the chief cause of death.

In a group of 7 interesting cases [17] the infection, after producing osteomyelitic destruction of the bone, extended into the soft tissue at the base of the skull and appeared in the face or neck. Meningitis was present in only 5 of these cases.

Osteomyelitis of the sphenoid may arise in a retrograde manner from cavernous sinus thrombosis due to other causes. This is due to the fact that the communicating veins are valveless and blood flow can occur in either direction. Faulkner mentioned four pathways by which cavernous sinus thrombosis may occur: (1) anteriorly, through the angular, supraorbital or supratrochlear veins; (2) internally, through the ethmoid or sphenoid veins; (3) inferiorly, through the pterygoid or carotid plexuses, and (4) posteriorly, through the petrosal veins. The literature contains reports of osteomyelitis of the sphenoid which developed in such a retrograde manner from the cavernous sinus, thrombosis occurring by all four routes.

Illustrative of development along the first pathway are the cases of Paunz and of Turner and Reynolds (1926 [*b*]), in both of which infection originated about the tip of the nose. In the case reported by Turner and Reynolds, early abscess of the frontal lobe was also found.

---

15. Pekosławsky, Schlagenhaufer, Barnes and Hilfrich.

16. Hawkins (case 9), Kirkland, Bronner, Finlay, Whitehead, Thomson (case 2), Stucky (case 14, 1911), Dean, Fink and Eagleton (1926, 2 cases).

17. Scholz, Panas (1873), Leber (case 4), Sandford, Dmochowski, Grünwald (1902) and Schröder.

The second route has already been considered. The third pathway was followed in 13 cases,[18] in all but 1 of which the infection originated about the tonsil. Ulcerative or gangrenous tonsillitis was present in 6 and a peritonsillar abscess in 5. In 1 case the infection followed removal of the tonsils and adenoids. In the case not due to tonsillar disease (Moreau), an abscess originated in the jaw and spread to the cervical region. In every case but 1, meningitis was present, and in all cases suppurative osteomyelitis of the sphenoid was seen.

The fourth route was followed in 3 cases,[19] in all of which osteomyelitis of the petrosa led to osteomyelitis of the sphenoid, meningitis and death.

Lying, as it does, directly over the sphenoid sinus, with the mucosa of which the sella is in communication, the pituitary gland is frequently involved in suppurations of this region. In many instances in the present series the pituitary gland was found bathed in pus, and in 5 cases [20] swelling or softening of the gland was noted. In 10 cases [21] suppuration or complete destruction of the gland was found, as it was in 6 others in which meningitis did not occur.[22] In another case (Pickworth, 1928) the pituitary gland was surrounded by a thick, ragged capsule. In case 1 of the present report the pituitary gland was reduced to a suppurative mass.

Disturbances of pituitary function from disease of the sphenoid sinus are rare but do occur. Meyer reported a case of adiposogenital syndrome from this cause, in which the testes showed aspermatogenesis and the pituitary gland was a suppurative mass. Torrigiani and Caliceti reported cases of pituitary syndrome and Ferreri a case of acromegaly, in all of which there was improvement after operation on the infected sphenoid sinus.

Glycosuria which could result from involvement either of the pituitary body or of the hypothalamic region has been reported in only 2 cases,[23] to the best of my knowledge. Since this condition occurred in 2 of my cases and in 1 other case, it seems worth while to report them here.

---

18. Panas (1885), Mitvalsky (case 1), Vossius (p. 16), Moreau (p. 81), Jacques and Lucien, O'Malley, Wylie and Wingrave, Höston (case 2), Ramdohr (case 2), Eagleton (1926, case F, page 83), Blumenthal, Hesse (case 7) and Boemer.

19. Lawson (2 cases) and Hesse (case 5).

20. Weichselbaum (1881), Coupland, Schlagenhaufer, Imperatori and Grant.

21. Palletta, Andral, Raymond, Stoerk, Wertheim, Moreau, Meyer, Boggs and Winternitz, Wylie and Wingrave, and Turner and Reynolds (1928 [*b*]).

22. Leveque-Lasource, Heslop, Popham, Holmes (1896), Rodger (1922) and Fink.

23. Schech, and Boggs and Winternitz.

REPORT OF CASES

CASE 1.—H. B., a man aged 70, was admitted to St. Joseph's Mercy Hospital on Sept. 26, 1935, in coma. The history obtained from the son was that the patient had had a cold, with profuse nasal discharge, for about ten days. About a week before entry he began to complain of pain in the frontal region, and two days prior to entry he complained of pain in the teeth in the right upper jaw and the right ear. He consulted his family physician, who told him that he had slight redness in the throat and an infection in the right ear. The right drum was incised, but this did not relieve the pain. On the day prior to entry the patient again consulted his physician, stating that he was sleepy and could scarcely keep awake. Nothing new was found, and the patient was sent home. By 6 o'clock in the evening he became comatose and had been in this condition ever since. The family were unable to arouse him, but frequently he became violent and had to be restrained. He was seen by his physician on the morning of entry, and a diagnosis of mastoiditis was made. He was referred to the hospital for an emergency operation on the mastoid.

Past History.—Fifteen years previously the patient fell from the roof of a barn on his head. It is not known whether he had a fracture of the skull or not, but since that time he had been unable to turn his head to the right. He had had no convulsions and no other evidences of intracranial injury. Six months before entry he had an illness which was thought to be rheumatism, but no accurate diagnosis was made. Since that time he had been fairly well until the present illness and had carried on his usual occupation as a farmer. He told his family on several occasions that he thought there was something wrong, but he did not consult a physician.

Physical Examination.—The patient was a well developed, rather obese, senile man who lay in bed with his eyes closed. He did not respond to questions or to irritation but occasionally exhibited spontaneous restlessness and attempted to get out of bed. Two attendants were assigned to restrain him. The pupils were of medium size and equal and reacted to light. The nares contained dried pus. The septum was markedly deflected to the left, but there was no perforation or ulceration. The turbinates were congested, and pus was noted in the upper portion of the nose. It was impossible to examine the throat because the patient held his teeth tightly closed. Both external auditory canals contained impacted cerumen. After removal of the cerumen the left canal appeared to be fairly normal, although there was slight redness in the fundus of the canal. The right canal was acutely inflamed and presented over the posterior portion a swelling which probably represented the incision made by the family physician. The drum membrane was gray, but no cone of light could be seen. Diagnostic myringotomy revealed that the middle ear was normal. There was no swelling over the mastoid on either side. There were no enlarged glands in the neck. Definite rigidity was present. Examination of the chest revealed no bulging or retraction of the interspaces. The heart was normal in size, with a normal pulse rate. The heart sounds appeared to be normal. The blood pressure was 140 systolic and 70 diastolic. The lungs were resonant throughout, and there were no rales. The abdomen was normal except for distention of the bladder. Catheterization released about 1 liter of dark brownish yellow urine, which on examination revealed a small amount of albumin, sugar (2 plus) and acetone (3 plus).

After making a neurologic examination Dr. H. W. Riggs said he believed that the patient was suffering from diabetic coma but that there was also advanced cerebral sclerosis. The patient was referred to Dr. Mark Marshall for treatment

of diabetes. Laboratory examination revealed: hemoglobin, 104 per cent; red blood cells, 3,420,000; white blood cells, 4,950; lymphocytes, 33 per cent; polymorphonuclears, 56 per cent, and band cells, 11 per cent. The blood sugar level was 200 mg. per hundred cubic centimeters and the urea level 34 mg. The Kahn test gave a negative reaction.

Diagnosis.—The diagnosis was coma, probably due to diabetes and cerebral sclerosis; mild nephritis; acute external otitis on the right (bilateral impacted cerumen was removed); deflection of the septum, and acute bilateral suppurative sphenoethmoid sinusitis.

Treatment.—The patient was given 5 per cent dextrose intravenously with insulin. This was repeated several times, with the practical disappearance of sugar from the urine. However, the patient gradually became worse and evidences of meningitis appeared. The pulse became more rapid and weak, and cyanosis appeared during the night. The patient died the following morning.

Postmortem Examination.—Autopsy revealed no striking abnormality except in the head. On removal of the calvarium, a purulent exudate was seen extending over the entire vertex and involving both hemispheres. It was more pronounced anteriorly over the frontal lobes, where there was considerable hemorrhagic extravasation. There was relatively little exudate over the lateral portion, and there was no evidence of infection from either ear. The right anterior and middle fossae were clear, but on the left side there was considerable exudate. The dura over the left sphenoid sinus and the pituitary gland was markedly inflamed, and the pituitary gland was almost completely destroyed by the inflammatory process. Dissection of the sinuses revealed a thick, edematous membrane in the left ethmoid sinuses, and the left sphenoid sinus was filled with purulent material. No evidence of osseous destruction was noted. The cavernous sinus had a suppurative thrombus. When the brain was opened no abnormality was noted except moderate vascular congestion. The ventricular fluid was definitely cloudy. Smears and cultures of material from the sphenoid sinus and the brain cavity revealed nonhemolytic streptococci.

Final Diagnosis.—The final diagnosis was acute purulent meningitis originating in an acute streptococcic sphenoiditis on the left, diabetes and nephritis.

CASE 2.—W. D., a man aged 54, was delirious. About a week before admission to the hospital he had an acute infection of the upper respiratory tract. He paid no attention to this and continued to work. Three days prior to admission to the hospital he began to note severe pain in the right ear. This ruptured spontaneously the following day. He began at that time to complain of a peculiar feeling in his head and told his wife that he had headache in the frontal region. Even at that time he continued working and refused the advice of his employer that he go home and go to bed. He had chills and fever but apparently paid no attention to these. On the following day, which was the day prior to his admission to the hospital, he began to be delirious. The family physician was called in and administered opiates. As it was felt that the right ear might be responsible for the condition, an otologist was consulted. Apparently a diagnosis of otitis media was made, and myringotomy was advised. The patient was then given chloroform, and the drum was incised. There was little change in the general condition from that time until the time of admission to the hospital. He quieted down considerably, however, but this was believed to be due to the opiate.

Past History.—The patient had always been in good health and had always worked hard. He had had the usual diseases of childhood but no serious illnesses or operations. His wife stated that he had some sort of heart attack about five

years before entry, but there was no repetition of this trouble. He had complained of slight dizzy spells during the past three or four years, but these had never incapacitated him for work. He had been subject to rather frequent head colds during the past six or seven years, during which attacks he usually had pain in the frontal region.

Physical Examination.—The patient was well developed and well nourished. He appeared to be acutely ill. He did not respond to questioning and resisted any attempt at examination. He seemed rather sleepy and did not show any signs of violence. There was slight pallor, and the lips were slightly cyanotic. The temperature was 102 F., the pulse rate 100, the respiratory rate 20 and the blood pressure 116 systolic and 72 diastolic. The nares contained crusted pus. The septum was rather markedly deflected to the left, but there was no ulceration or perforation. The turbinates were considerably swollen, and there was pus in the sphenoethmoid region on both sides. The mouth was edentulous. The tongue was heavily coated. The tonsils were enlarged and septic. Pus was present on the posterior wall of the pharynx. There were a few small glands in the neck, but no abscess formation was apparent. The left external auditory canal was clean, and the tympanic membrane was normal. The right external canal contained a small amount of serosanguineous fluid. The drum was reddened, and there appeared to be a small perforation in the inferior posterior quadrant. No bulging was apparent, and the short process was visible. The chest was essentially normal with the exception of a few rales at the bases of both lungs. Neurologic examination revealed that the Brudzinski sign was present; the Kernig sign was not elicited. The neck and back were stiff. The reflexes were hyperactive, and there was no ankle clonus. Lumbar puncture revealed a cloudy fluid under no increase in pressure. A smear revealed large numbers of gram-positive diplococci.

Diagnosis.—The diagnosis was purulent meningitis, probably pneumococcic; acute bilateral suppurative sphenoethmoiditis; deviation of the septum, and acute faucial tonsillitis.

Course.—Treatment was not given, as the condition was considered hopeless. The patient died about eighteen hours after examination. Permission for an autopsy could not be obtained.

Comment.—A third case belonging to this group was that of a patient in the service of Dr. George F. Muehlig, who gave me permission to report it.

CASE 3.—Mrs. C. V., aged 64, was admitted on Nov. 16, 1932. Twenty years previously she had an operation on the left mastoid, performed by Dr. R. B. Canfield. She recovered completely and was well until two years before the present admission to the hospital, when she underwent another operation for drainage of the gallbladder and appendicectomy. She recovered and was well until about a week prior to the present admission to the hospital, when she was taken with an acute infection of the upper respiratory tract. She had no fever but felt sick enough to remain in bed part of the time. On the day prior to admission to the hospital the temperature went up to 101 F., and she complained of severe headache, nausea and vomiting. The following morning she became delirious and was brought to the hospital.

Physical Examination.—Examination revealed a well developed woman in coma. The left upper lid was ptotic, but the right lids were normal. The pupils reacted to light and in accommodation. There was some stiffness of the neck, but the Kernig sign was not elicited. The knee jerks were normal. The heart and lungs

appeared to be essentially normal for a person of her age. The urine showed albumin (3 plus) and a trace of sugar. The white blood cell count was 29,800. Lumbar puncture revealed cloudy fluid under pressure. The cell count of the fluid was 17,500, but no organisms were found. Culture, however, revealed pneumococci. At about 9:30 that evening the respirations became noisy, the lungs began to fill up with fluid and the heart became weak and rapid. Death occurred about four hours later.

Postmortem Examination.—Autopsy, by Dr. S. C. Howard, revealed a diffuse purulent exudate over the hemispheres, with a marked accumulation at the base of the brain. All the pial vessels were engorged, and the brain was edematous but showed no localized suppuration or necrosis. The superior longitudinal sinus showed an old thrombosis but no recent involvement. The dura in the region of the old mastoid operation appeared to be thickened, but there was no lesion of continuity. The upper portion of the left petrosa was thin, and after its removal, osseous destruction with old granulations was noted near the labyrinth. There was no evidence of recent activity there, and pus was not seen. The ethmoid and sphenoid sinuses, however, showed an active infection and were filled with purulent material.

It was the opinion of the pathologist and of the otologic consultant, Dr. A. C. Furstenberg, that the meningitis originated in the sinusitis and not in the older process in the temporal bone.

Comment.—In none of these 3 cases had glycosuria been present prior to the onset of the present illness, and it is conceivable that it could have been due either to involvement of the pituitary gland, as was demonstrated in the first case, or to involvement of the floor of the third ventricle by the meningitis.

A fourth case of meningitis from the sphenoid sinus in which glycosuria was not exhibited occurred in the service of Dr. A. C. Furstenberg, who gave me permission to report it here.

CASE 4.—D. W., a boy aged 16, was admitted on Aug. 10, 1931. About a week prior to entry he went swimming and afterward began to note marked nasal obstruction and discharge. Two days before entry the left eye and the left side of the face began to swell, and he complained of acute pain over the left eye. He was treated conservatively at home, but as the swelling increased he was brought to the hospital.

Physical Examination.—Examination revealed a sick boy, with marked swelling over the entire forehead, extending well up into the hair line. The left eye was swollen shut, and there was great swelling over the cheek and parotid region. There was tenderness over the parotid region. With the use of a local anesthetic an incision, 1 inch (2.5 cm.) long was made over the left brow, and a considerable amount of dirty gray sanguineous purulent material escaped. The temperature, which was around 104 F., dropped a little after this procedure, but the next day it went back to 104 F., and there was proptosis of both eyes. The patient was profoundly septic and showed some rigidity of the neck, but the Kernig sign was not elicited. Two days later he died.

Postmortem Examination.—Autopsy, performed by Dr. S. C. Howard, revealed widespread edema of the head, which increased the thickness of the scalp to about 4 cm. The scalp showed marked necrosis throughout but little frank pus. When the calvarium was removed, areas of exudate were noted over the external surface

of the brain, with only a slight amount of exudate at the base. The superior longitudinal, left cavernous, left lateral and sigmoid sinuses showed purulent thrombosis. The brain showed no localized areas of infarction or softening. The pituitary body was small but showed marked congestion. The sphenoid sinuses were filled with purulent gelatinous material, as were both maxillary and frontal sinuses. The mastoids were free from any recognizable pathologic changes. The opinion of the pathologist was that the infection in the sphenoid sinus had produced thrombosis of the cavernous sinus on the left and that this, in turn, had given rise to the meningitis.

COMMENT

The association of sphenoid disease and insanity has been noted by many authors. In a fairly large group of cases [24] the mental aberration improved or disappeared after drainage of the infected sphenoid sinus. Graves examined the sinuses of 1,001 patients with mental disorders clinically by means of the Watson-Williams technic and found that 818 had sinus disease; 504 showed involvement of the sphenoid sinus, 424 of the ethmoid sinus and 631 of the antrum. Treatment was followed by improvement in nearly all cases. Of the entire group, 485 were able to be discharged to the care of friends, and 300 others were improved but not enough to be discharged. The article includes an excellent review of the relation of sinusitis to many abnormal mental states, muscle tone and sensory, cutaneous, circulatory, pulmonary and gastrointestinal disturbances; it well repays one to read the article in full.

Pickworth (1932), who worked with Graves as pathologist, noted abnormally large amounts of colloid in the pituitary body in 3 cases, in 2 of which the anterior and posterior lobes were separated by this material. Pickworth also studied 5 other cases (8 in all) in which the only or the chief cause of death was infection in the sphenoid sinus, with more or less abnormality of the pituitary body. Mott and Robertson examined the pituitary body histologically in 72 cases of insanity and observed changes in nearly all. There was a marked degree of exhaustion of the cellular elements, amounting in some cases to almost complete atrophy. Robertson reported a case of periodic insanity in which the chief cause of death was necrosis of the posterior wall of the sphenoid sinus with an extradural abscess.

In discussing the etiologic relation of disease of the sphenoid sinus to insanity, Pickworth cited the work of Orr and Rows, who placed a capsule containing Staphylococcus aureus in contact with the carotid artery in the neck and noted hyaline thrombosis of vessels in various parts of the brain, along with coagulation necrosis of the nerve cells in certain areas. He reasoned that the finding of bacteria in and about

24. Wright, Stucky (1906), Sharp, Faulkner (1921), Hubert, Gatewood, Watson-Williams, Eden and Yates, French and Mithoefer.

the walls of the vessels passing near the sphenoid sinus could duplicate this and could act as a starting point for changes in the brain. At all events, the work of Graves has indicated that there is hope for many persons afflicted with insanity whose condition has heretofore been considered hopeless.

There is complete unanimity among authors that when infection extends beyond the sphenoid sinus into the orbit, blood spaces or brain, the prognosis is extremely grave. However, a study of the literature reveals that in at least 7 cases recovery has followed a surgical attack on the sphenoid sinus. Ridpath stated that he had never seen recovery in such a case.

Kander, in 1907, noted a case in which there were empyema of the sphenoid sinus and cocci in the spinal fluid. Opening of the empyema was followed by recovery. Avellis (1907) studied a case in which spontaneous recovery occurred. Eagleton (1932 [*b*]) reported a case in which surgical treatment of the ear and sphenoid sinus, combined with the use of pneumococcus (type II) serum, was followed by recovery. Kramer and Som (1936) noted 4 recoveries in 19 cases; and Smith (1936), 1 recovery in 3 cases.

Kramer and Som advocated first intranasal sphenoethmoidectomy. If the symptoms persisted, they then stripped the mucosa and investigated the basisphenoid with curets, chisels and other instruments. In 1 of their cases they found a purulent collection in the bone. All the patients with bacterial meningitis died, as did all of those with an initial spinal fluid count over 500.

Smith advised opening through the basisphenoid with a burr, incising the dura and inserting a tube into the basal cistern. He advocated the use of intratracheal anesthesia and an approach through the mouth.

Eagleton (1921, 1924) advised, in addition, ligation of the internal carotid artery to put the parts at rest, repeated transfusions from an immunized donor, specific serums when available and massive lumbar punctures.

With the recent increase of interest in this subject and with the knowledge of the symptomatology and associated pathologic changes in disease of the sphenoid sinus, it is not too much to hope that the next few years will show a decrease in the deaths from this cause, not so much from treatment but from prevention by early attack on the diseased sphenoid sinus.

SUMMARY

A historical review of the reported cases of meningitis from the sphenoid sinus is presented, along with abstracts of the reports of some of the first cases, most of which are not included in previous monographs on the subject. Statistical reports are studied, and the conclu-

sion is drawn that while the sphenoid sinus is involved in about 15 per cent of clinical cases of sinusitis and in 33 per cent of pathologic cases, it is nevertheless responsible for approximately 35 per cent of all rhinogenous intracranial complications. Evidence is presented which indicates that the spread to the meninges and venous spaces is predominantly vascular and that this extension is aided by the close anatomic relation of the vascular marrow spaces of the sphenoid bone with the infected mucosa of the sinus.

From the literature are gathered reports of 129 cases of meningitis from the sphenoid sinus. There were in this group 7 instances of extradural abscess, 7 of cerebral and 1 of cerebellar abscess; 6 of suppurative and 1 of hemorrhagic encephalitis; 20 cases in which there was concomitant otitis and 47 in which there was purulent thrombosis of the cavernous sinus, either alone or with thrombosis of communicating venous spaces of the head. In 4 cases there was thrombosis of the superior longitudinal sinus, and in 2 cases the internal carotid artery was thrombosed within the diseased cavernous sinus. A number of cases are noted in which infection of the sphenoid sinus and meningitis arose in a retrograde manner from the cavernous sinus which had become diseased by extension of infection from other portions of the head.

The relation of the sphenoid sinus to the pituitary body is discussed, and cases of pituitary dysfunction due to disease of the sphenoid sinus is noted. Since glycosuria was noted in only 2 of the cases reported in the literature, 3 additional cases are recorded in which this finding was made. The further relation between disease of the sphenoid sinus and of the pituitary body and insanity is noted, with evidence that in certain cases of mental aberration, resolution of the infection within the sphenoid sinus is remedial.

Treatment is finally discussed. One case is noted in which spontaneous recovery occurred, and in 6 others, recovery followed surgical attack on the sphenoid sinus. The methods of Eagleton and of Smith are mentioned particularly, and the hope is expressed that the future management of these cases of serious involvement will be more radical and successful.

NOTE.—Since this article was prepared for publication an additional case of recovery from localized meningitis after operation on the infected sphenoid sinus has been noted (Shambaugh, G. E., Jr.: The Surgical Treatment of Meningitis of Otitic and Nasal Origin, *J. A. M. A.* **108:** 696 [Feb. 27] 1937). The patient was well for three years and then apparently had an exacerbation of the disease and died. Autopsy was not permitted.

410 Highland Road.

BIBLIOGRAPHY

Abercrombie, J.: Pathological and Practical Researches on Diseases of the Brain and the Spinal Cord, Philadelphia, Lea & Blanchard, 1843, p. 33.

Andral, G.: Maladies de l'encéphale, in Clinique médicale, Paris, Deville Cavellin, 1834, vol. 5, p. 39.

Andrews, A. H., in discussion on Yerger (1921).

Avellis, G.: Radiogrammes d'un cas de cancer primitif du sinus sphénoïdal, Presse oto-laryng. belge **4**:475, 1905.

Oertliche seröse Meningitis bei akuter Keilbeineiterung mit Spontanheilung, Ztschr. f. Ohrenh. **54**:403, 1907.

Barnes, S.: Septic Meningitis Secondary to Disease of the Nose, Birmingham M. Rev. **6**:533 (Sept.) 1905.

Beck, J. C., and Guttmann, M. R.: Basaloma or So-Called Cylindroma of the Air-Passages [case 3], Ann. Otol., Rhin. & Laryng. **45**:618 (Sept.) 1936.

Beek: Russk. vrach, 1903, p. 574; abstracted, Rev. hebd. de laryng. **23**:265, 1903.

Beevor, C. E., and Buzzard, E. F.: Case of Basal Meningitis Due to Streptothrix Infection, Tr. Path. Soc. London **54**:320, 1903.

Bergengrün, P.: Ueber einen letalen Fall von Empyema Sinus ethmoidalis, St. Petersburg. med. Wchnschr. **30**:53 (Feb. 12) 1905.

Berger, E., and Tyrman, J.: Die Krankheiten der Keilbein-Höhle und des Siebbein-Labyrinthes und ihre Beziehungen zu Erkrankungen des Sehorgans, Wiesbaden, J. F. Bergmann, 1886.

Bertemes, G.: Etude anatomo-topographique du sinus sphénoïdal; application à la pathologie des sinusites sphénoïdales, Nancy, A. Crépin-Leblond, 1900.

Birch-Hirschfeld, A.: Die entzündlichen Erkrankungen der Orbita, in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Englemann, 1909, vol. 9, pt. 2, chap. 13, p. 304.

Blumenthal, B.: Osteomyelitis des grossen Keilbeinflügels, Ztschr. f. Laryng., Rhin., Otol. **17**:335, 1929.

Boemer, L. C.: The Great Vessels in Deep Infection of the Neck, Arch. Otolaryng. **25**:465 (April) 1937.

Boggs, T. R., and Winternitz, M. C.: Acute Suppurative Hypophysitis as a Complication of Purulent Sphenoidal Sinusitis, Johns Hopkins Hosp. Rep. **18**:56, 1919.

Bombelli, U.: Un caso di ascesso cerebrale da sinusite sfenoidale latente in paziente affetta da gravi complicanze otitiche, Riv. oto-neuro-oftal. **5**:142 (March-April) 1928.

Bondy, G., in discussion on Neumann.

Braun, H.: Ueber totale doppelte Oberkieferresectionen (case 2), Arch. f. klin. Chir. **19**:728, 1876.

Broeckaert, J.: Un cas de mort à la suite d'une opération de polyps de nez, Ann. d. mal. de l'oreille, du larynx **20**:1288, 1894.

Bronner, A.: Notes on a Case of Thrombosis of the Cavernous Sinus Due to Empyema of the Sphenoidal Sinus, Brit. M. J. **2**:1310 (Nov. 12) 1904.

Burger, H.: Die endokraniellen Komplikationen der Nasennebenhöhlenentzündung, in Denker, A., and Kahler, O.: Handbuch der Hals- Nasen- Ohrenheilkunde, Berlin, Julius Springer, 1926, vol. 2, p. 898.

Caliceti, P.: Syndrome oculo-hypophysaire par sinusite sphénoïdale purulente chronique, Rev. de laryng. **46**:13, 1925.

Campbell, E. H.: The Cavernous Sinus: Anatomical and Clinical Considerations, Ann. Otol., Rhin. & Laryng. **42**:51 (March) 1933.

Cange, A.: L'ostéomyélite aiguë des petites ailes du sphénoïde, Arch. d'opht. **39**:321 (June) 1921.

Caubet, H., and Druault, A.: Méningite et phlegmon de l'orbite dus à une polysinusite d'origine dentaire, Ann. d. mal. de l'oreille, du larynx **25**:211, 1899.

Cavanaugh, J. A.: The Sphenoid on Parade, Laryngoscope **45**:911 (Dec.) 1935.

Chisholm, J. J., and Watkins, S. S.: Twelve Cases of Thrombosis of the Cavernous Sinus, from a Study of Fifty Thousand Surgical Histories in the Johns Hopkins Hospital [case 5], Arch. Surg. **1**:483 (Nov.) 1920.

Coupland, S.: On a Case of Ophthalmoplegia Dependent upon Thrombosis of the Cavernous Sinuses, Tr. Ophth. Soc. U. Kingdom **7**:228, 1887.

Cullom, M. M.: The Chronicity of Sinus Disease and Its Relation to Middle Ear Infection, Deafness and Constitutional Disease, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1936, pp. 84-106.

Davison, C., and Kahr, S.: Cancer of the Sphenoid Sinus with Invasion of the Cranial Cavity, Arch. Otolaryng. **14**:16 (July) 1931.

Dean, L. W., in discussion on Langworthy, H. G.: Cavernous Sinus, Ann. Otol., Rhin. & Laryng. **25**:554 (Sept.) 1916.

Demarquay: Polype glandulaire de la muqueuse nasale: Repullution extirpation et cauterisation; accidents consecutifs a l'opération; paralysie de tous les muscles de l'oeil; symptomes d'empoisonnement par la morphine; mort; autopsie, Gaz. d. hôp. **42**:277 (June 22) 1869.

Dmochowski, Z.: Beitrag zur pathologischen Anatomie und Aetiologie der entzündlichen Processe im Antrum Highmori, [case 25], Arch. f. Laryng. u. Rhin. **3**:255, 1895.

Dodd, O.: Abscess of the Sphenoidal Sinus Occurring with Acute Mastoiditis, Laryngoscope **10**:334 (May) 1901.

Douglas, B.: Die Beziehung der Siebbeinzellen zur Keilbeinhöhle mit besonderer Berücksichtigung des Sinus im kleinen Keilbeinflügel, Monatschr. f. Ohrenh. **35**:392 (Sept.) 1901.

Dreyfuss, R.: Die Krankheiten des Gehirns und seiner Adnexa im Gefolge von Naseneiterungen, Jena, Gustav Fischer, 1896.

Druss, J. G.: Intracranial Complications Associated with Disease of the Sphenoid Bone. Report of Two Cases: 1. Meningitis Secondary to Infection of the Craniopharyngeal Canal (?) and 2. Abscess of the Frontal Lobe, Arch. Otolaryng. **10**:39 (July) 1929.

Duplay, S.: Ozène et otite purulente probablement de nature syphilitique; phlébite des sinus du crâne et de la veine ophtalmique; méningite purulente; mort, Arch. gén. de méd. **2**:348 (Sept.) 1874; cited by de Lapersonne: Phlébite suppurée des veines ophtalmiques et des sinus caverneux, Arch. d'opht. **5**: 436, 1885.

Eagleton, W. P.: The Operative Treatment of Suppurative Meningitis with Special Reference to Irrigation of the Cranial and Spinal Subarachnoid Spaces, and the Importance of Protective Meningitis from a Prognostic and Therapeutic Standpoint, with an Analysis of the Cases of Recovery—Exclusive of Meningococcic—Reported in the Literature, Tr. Am. Otol. Soc. **15**:227, 1921.

The Surgical Treatment of Meningitis, J. A. M. A. **83**:1900 (Dec. 13) 1924.

Cavernous Sinus Thrombophlebitis and Allied Septic and Traumatic Lesions of the Basal Venous Sinuses, New York, The Macmillan Company, 1926, p. 148.

The Carotid Venous Plexus as the Path of Infection in Thrombophlebitis of the Cavernous Sinus: Its Relation to Retropharyngeal and Sphenoidal Suppuration, Arch. Surg. **15**:275 (Aug.) 1927.

(*a*) Suppurative Meningitis of Otitic and Nasal Origin: Its Relation to Blood Stream Invasion of the Pial Vessels, Arch. Otolaryng. **15**:884 (June) 1932.

(*b*) Meningitis from the Sphenoid, Tr. Am. Laryng., Rhin. & Otol. Soc. **38**:51, 1932.

Meningitis from the Petrous Apex and the Sphenoidal Basis, J. M. Soc. New Jersey **32**:125 (March) 1935.

Eden, G., and Yates, A. L.: Treatment of Encephalitis Lethargica by the Removal of Possible Etiologic Factors, Brit. M. J. **1**:714 (April 16) 1927.

Embleton, D.: Sphenoidal Empyema and Epidemic Cerebro-Spinal Fever, Brit. M. J. **1**:7 (Jan. 3) 1920.

——and Peters, E. A.: Cerebro-Spinal Fever and the Sphenoidal Sinus, Lancet **1**:1078 (May 22) 1915.

Emerson, F. P.: Developmental Absence of the Outer Right Sphenoidal Wall, Occupied by a Vein Communicating Directly with the Cavernous Sinus: Operative Fatality and Autopsy, Ann. Otol., Rhin. & Laryng. **17**:479 (June) 1908.

Fallopius, G.: Observationes anatomicae, Venice, Marcum Antonium Ulmum, 1561, p. 32.

Faulkner, E. R.: Sphenoidal Sinusitis with Marked Cerebral Symptoms; Operation; Recovery, Laryngoscope **31**:232, 1921.

Rhinological Aspects of Cavernous Sinus Thrombosis, Surg., Gynec. & Obst. **52**:474 (Feb. 15) 1931.

Ferreri, G.: Disturbi ipofisari guariti con la trapanazione del seno sfenoidale, Riv. oto-neuro-oftal. **4**:21 (Jan.-Feb.) 1927.

Field, C. E., and Rogers, H.: A Fatal Case of Meningitis Due to Infection of the Sphenoid Sinus by Pfeiffer's Bacillus, with a Short Review of Methods of Treatment, Bristol Med.-Chir. J. **53**:151, 1936.

Fink, E. B.: The Intracranial Complications of Posterior Sinus Infections: Report of an Unusual Case with Autopsy Findings, Surg., Gynec. & Obst. **41**:589 (Nov.) 1925.

Finlay, C. E.: Ueber einen Fall von Thrombophlebitis des Sinus cavernosus kompliziert durch Empyem der Keilbeinhöhlen und der Siebbeinzellen, irrtümlicherweise für eine Thrombophlebitis des Sinus lateralis gehalten, Ztschr. f. Ohrenh. **48**:227, 1904; Arch. Otol. **32**:419, 1903.

Flatau, T. S.: Un caso di carie delle pareti del seno sfenoidale, Arch. ital. di laring. **13**:26, 1893; in discussion on Herzfeld, J.: Ueber Empyem Resp. Caries der Keilbeinhöhle, Berl. klin. Wchnschr. **32**:901 (Oct. 14) 1895.

Forget, A.: Polype fibreux occupant les fosses nasales, le sinus maxillaire et le pharynx, Union méd., Paris **4**:587 and 599, 1850.

Foucher, A. A.: Necrose du sphénoïde, Rev. internat. de rhin., otol. & laryng. **16**:301, 1896.

Fraenkel, E.: Beiträge zur Pathologie und Aetiologie der Nasennebenhöhlen-Erkrankungen, Virchows Arch. f. path. Anat. **143**:42, 1896.

Fraser, J. S.: Zwei Fälle von Nebenhöhleneiterung mit tödlicher Komplikation, Internat. Centralbl. f. Laryng., Rhin. **37**:416, 1921.

The Frequency of Sphenoidal Operations, J. Laryng. & Otol. **39**:393 (July) 1924.

Fremel, F.: Ueber Meningitis nach Keilbeinhöhlen- und Mittelohreiterung, Monatschr. f. Ohrenh. **56**:230 (April) 1922.

French, J. G.: Infection of the Nasal Sinuses in Relation to Insanity, Lancet **2**: 13 (July 2) 1927.

Furstenberg, A. C.: Osteomyelitis of the Skull: The Osteogenic Processes in the Repair of Cranial Defects, Ann. Otol., Rhin. & Laryng. **40**:996 (Dec.) 1931.

The Pathology of the Spread of Osteomyelitis of the Skull: Its Relation to Brain Abscess, Tr. Am. Laryng., Rhin. & Otol. Soc. **39**:423, 1933.

Gallemaerts: Bull. Soc. belge d'opht., December 1899, p. 29; Ann. d'ocul. **123**: 142, 1900.

Galtung: Fall von Thrombose im Sinus cavernosus von der Keilbeinhöhle aus, Internat. Centralbl. f. Laryng., Rhin. **37**:181, 1921.

Gatewood, E. T.: Acute Toxic Encephalitis of Nasal Sinus Origin: Report of Two Operative Cases with Recovery, J. A. M. A. **84**:1473 (May 16) 1925.

Gerber: Meningitis nach larvierter Nebenhöhleneiterung, Ztschr. f. Ohrenh. **63**: 150, 1911.

Germann, T.: Zur Symptomatologie, Therapie, und Prognose der orbitalen Augenerkrankungen, veranlasst durch Empyeme der Nebenhöhle der Nase, Mitt. a. d. St. Petersburg. Augenh. **5**:38, 1898.

Gowers, W. R.: A Manual of Diseases of the Nervous System, London, J. & A. Churchill, 1893, vol. 2, p. 470.

Grant, A. L.: Acute Abscess in the Body of the Sphenoid, Ruptured into the Pituitary Fossa: Autopsy Report, Laryngoscope **41**:842 (Dec.) 1931.

Graves, T. C.: Sinusitis in Mental Disorder: I. Clinical Manifestations and Illustrative Cases [case 29], J. Ment. Sc. **78**:459 (July) 1932.

Greene, D. W.: A Case of Unrecognized Empyema of the Right Sphenoidal Sinus; Great Exophthalmos and Divergence of Right Eyeball; Extensive Edema of Right Side of Face and Neck; Death; Autopsy, Ophth. Rec. **6**: 346 (July) 1897.

Grünwald, L.: A Treatise on Nasal Suppuration, translated by W. Lamb, London, Baillière, Tindall & Cox, 1900, p. 140.

Intervertebraler Abscess, ausgehend von einer Eiterung einer accessorischen Keilbeinhöhle; zahlreiche Complicationen, Arch. f. Laryng. u. Rhin. **12**:454, 1902.

Guibert: Anévrisme artériel de la carotide interne au niveau du sinus caverneux gauche: Communication avec le sinus sphénoïdal droit; hemorrhagies nasales; mort; autopsie, Ann. d'ocul. **113**:314, 1895.

Hadjopoulos, L. G., and Bell, J. W.: Direct Versus Intermediate Pathways in Infections of the Mastoid, Arch. Otolaryng. **25**:601 (June) 1937.

Hajek, M.: Pathologie und Therapie der entzündlichen Erkrankungen der Nebenhöhlen der Nase, ed. 4, Leipzig, Franz Deuticke, 1915, p. 438.

Halász, H.: Ein Fall von Encephalitis in Folge von Eiterung im Antrum sphenoïdale, Monatschr. f. Ohrenh. **39**:422 (Sept.) 1905.

Halle: Demonstration von drei Präparaten, Berl. klin. Wchnschr. **39**:506 (May 26) 1902.

Handke, F.: Lymphosarkom der Keilbeinhöhlenschleimhaut unter dem Bilde einer rhinogenen Meningitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:44, 1936.

Harke, E. A. T.: Beiträge zur Pathologie und Therapie der oberen Athmungswege, Wiesbaden, J. F. Bergmann, 1895.

Hawkins, H. P.: The Diagnosis and Treatment of the Pyemic Complications of Ear Disease [case 9], St. Thomas's Hosp. Rep. **18**:117, 1888.

Heslop, T. P.: Case of Hypertrophy with Abscess of the Pituitary Body, Dublin Quart. J. M. Sc. **6**:467, 1848.

Hesse, W.: Zur Aetiologie der Keilbein-Osteomyelitis, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:94 (Nov.) 1933.

Hilfrich, K. J.: Ueber intraorbitale Komplikationen bei akuten und chronischen Nebenhöhlenaffektionen [case 3], Ztschr. f. Ohrenh. **70**:31, 1914.

Häggström, A.: abstracted, Ztschr. f. Ohrenh. **70**:93, 1914.

Höston, K.: Om trombose av sinus cavernosus, Norsk mag. f. lægevidensk. **82**: 559 (Aug.) 1921.

Holmes, C. R.: The Sphenoidal Cavity and Its Relation to the Eye, with Ten Illustrations and Tabulated Measurements of Fifty Specimens from the Author's Collection, Arch. Ophth. **25**:460, 1896.

Holmes, E. M.: Suppuration of the Frontal, Ethmoid, and Sphenoid Sinuses, Boston M. & S. J. **148**:310 (March 19) 1903.

Horner, F.: Periostitis Orbitae und Perineuritis n. opt., Klin. Monatsbl. f. Augenh. **1**:71, 1863.

Hubby, L. M.: A Case of Internal Pachymeningitis, Leptomeningitis, Pansinusitis, Cavernous and Lateral Sinus Thrombosis, Laryngoscope **31**:204 (March) 1921.

Hubert, L.: Sphenoidal Sinusitis with Marked Cerebral Symptoms; Operation; Recovery, Laryngoscope **31**:322, 1921.

Huizinga, E.: Geneesk. gids **3**:561, 1925; cited by Eagleton, W. P.: Intradural Complications of Aural and Nasal Origin: Survey of Recent Literature, Arch. Otolaryng. **4**:69 (July) 1926.

Hyrtl, J.: Handbuch der topographischen Anatomie, Vienna, W. Braumüller, 1853, vol. 1, p. 192.

Imperatori, C. J.: Report of a Case of Chronic Frontal Sinusitis, Sphenoiditis, Influenzal Meningitis; Death; Necropsy, Laryngoscope **25**:580 (Aug.) 1915.

Jacobsgaard, J.: Pathologisch-anatomische Beiträge zur Frage nach den Fortleitungswegen der rhinogenen Meningitis, Ztschr. f. Laryng., Rhin., Otol. **26**: 386, 1935.

Jacques and Lucien: Phlegmon périamygdalien mortel par thrombo-phlébite du sinus caverneux, Bull. et mém. Soc. franç. d'oto-rhino-laryng. **24**:146, 1908.

Jassniger, K.: Der pneumococcus Friedländer als Erreger der eitrigen Meningitis cerebro-spinalis, Centralbl. f. Bakt. (Abt. 1) **30**:1 (July 12) 1901.

Jessop, W. H.: Two Cases of Proptosis Associated with Disease of Ethmoid and Sphenoid, Respectively [case 2], Tr. Ophth. Soc. U. Kingdom **23**:177, 1903.

Kander, L.: Ueber die intracraniellen Erkrankungen beim Empyem der Keilbeinhöhle, Beitr. z. klin. Chir. **36**:266, 1902.

Meningitis beim Keilbeinhöhlenempyem mit Ausgang in Heilung, Ztschr. f. Ohrenh. **54**:403, 1907.

Kaplan, A.: Recovery from Abscess of the Frontal Lobe Secondary to Empyema of the Sphenoid Sinus, Arch. Otolaryng. **25**:66 (Jan.) 1937.

Kaufman, A., and Hartmere, S. J.: Latent Osteomyelitis of the Sphenoid Bone Reactivated by Trauma, with Death from Meningitis, Laryngoscope **44**:477 (June) 1934.

Key-Åberg, H.: Thrombosis of the Cavernous Sinus, Acta oto-laryng. **3**: 36, 1921.

Kicer, G.: Latent Empyema of the Accessory Cavities of the Nose, Laryngoscope **6**:81 (Feb.) 1899.

Killian, cited by Luc, H.: Complications crâniennes et intracrâniennes des antrites frontales suppurrées, Ann. d. mal. de l'orielle, du larynx **35**:265, 1909.

Kirkland, T. S.: The Pathological Conditions of the Cranial Sinuses, J. Laryng. & Otol. **17**:561 (Nov.) 1902.

Knutson, E., abstracted, Internat. Centralbl. f. Laryng., Rhin. **32**:360, 1916.

Koebel: Ueber Kombination von Otitis media mit rhinogenem Gehirnabscess, Beitr. z. klin. Chir. **25**:526, 1899.

Kramer, R.: Intracranial Complications Following Sphenoid Infection, Laryngoscope **39**:573 (Sept.) 1929.

——and Som, M. L.: Actinomycosis of the Sphenoid with Actinomycotic Meningitis and Brain Abscess, Ann. Otol., Rhin. & Laryng. **44**:973 (Dec.) 1935.

Sphenoiditis with Meningitic Symptoms, Laryngoscope **46**:507 (July) 1936.

Lawson, L. J.: Osteomyelitis of the Sphenoid Bone: A Report of Two Cases, Arch. Otolaryng. **25**:1 (Jan.) 1937.

Leber, T.: Klinische ophthalmologische Miscellen, Arch. f. Ophth. **26**:249, 1880.

Lebert, T.: Ueber Gehirnabscesse, Virchows Arch. f. path. Anat. **10**:383, 1856.

Leegaard, F.: On Cerebral Abscesses of the Frontal Lobe Issuing from the Frontal Sinus, and Other Intracranial Complications Resulting from Inflammatory Processes of the Nasal Accessory Sinuses, Acta oto-laryng. **1**:343, 1918-1919.

Leuch, O.: Osteomyelitis acuta des Os sphenoidale, Basel, B. Schwalbe & Co., 1920.

Leveque-Lasource, A.: Observation sur un amaurose et un cophose, avec perte ou diminution de la voix, des mouvements, etc., par suite de lésion organique apparente de plusiers parties du cerveau, J. gén. de méd. **37**:368, 1810.

Lichtwitz, L.: Complications des empyèmes des cavites accessoires du nez, Ann. d. mal. de l'oreille, du larynx **22**:113, 1896.

Lieutaud: Abcès des sinus frontaux sphénoïdaux et maxillaires; mort, Mém. Acad. roy. d. sc., 1735, p. 18; cited by Lichtwitz.

Lischkoff, M. A.: Latent Brain Abscess Secondary to Sphenoid Sinusitis, with Case Report, South. M. J. **28**:700 (Aug.) 1935.

Lloyd, J.: Case of Proptosis from Thrombosis of the Cavernous Sinuses; Aneurysms of the Internal Carotid and Basilar Arteries from Suppurative Periarteritis; Death; Post-Mortem Appearances, Ophth. Rev. **3**:325 (Nov.) 1884.

Lodge, S.: A Case of Fatal Sphenoidal Suppuration, Laryngoscope **8**:139 (March) 1900.

Three Fatal Cases of Sinus Suppuration [case 1], Brit. M. J. **2**:601 (Aug. 30) 1902.

Lubliner: Zwei anatomische Präparate, Internat. Centralbl. f. Laryng., Rhin. **29**: 480, 1913.

Empyema Sinus sphenoidalis und Meningitis, Monatschr. f. Ohrenh. **48**:1327, 1914.

Lyonnet, B., and Regaud, C.: Tumeur carcinomateuse de l'arrière cavité des fosses nasales; envahissement du sphénoïde; paralysie de tous les nerfs crâniens du côté gauche, sauf l'olfactif et l'optique; mort par méningite, Ann. d. mal. de l'oreille, du larynx **19**:197, 1893.

Maas, H.: Zur Casuistik und Therapie der Gehirnabscesse nach eigenen Erfahrungen, Berl. klin. Wchnschr. **6**:127 (March 29) 1869.

McBean, G. M.: Variations of Sphenoid Sinus Disease [case 1], Ann. Otol., Rhin. & Laryng. **23**:419 (June) 1914.

Macewen, W.: Pyogenic Infective Diseases of the Brain and Spinal Cord, Glasgow, J. Maclehose & Co., 1893, p. 251.

Mackenzie, W.: Practical Treatise on the Diseases of the Eye, Boston, Carter, Hendee & Co., 1833, p. 69.

McMahon, B. J.: The Relation of Mastoid Structure to Extension of Mastoid Infection, Ann. Otol., Rhin. & Laryng. **40**:53 (March) 1931.

Martin, J. M. L. F.: De la frequence de l'empyème des cavites accessoires du nez, Thesis, Bordeaux, no. 34, 1899.

Maxwell, D.: Analysis of the Statistical Tables for the Years 1907 to 1926 Inclusive, Covering a Period of Twenty Years, J. Laryng. & Otol. **44**:105 (Feb.) 1929.

Meurers: Ueber 4 im Anschluss an eitrige Nebenhöhlenerkrankungen entstandene Komplikationen mit tödlichem Ausgang [case 3], Ztschr. f. Ohrenh. **60**:335, 1910.

Meyer, O.: Demonstration einiger seltener Präparate von Hirntumor und Erkrankungen der Hypophysengegend [case 4], Allg. Ztschr. f. Psychiat. **74**:521, 1918.

Mithoefer, W.: Acute Sphenoid Sinus Infections, Ohio State M. J. **23**:296 (April) 1927.

Mitvalsky: Contribution à la connaissance de la thrombophlébite orbitaire, Arch. d'opht. **16**:22, 1896.

Möller, J., in discussion on Schousboe: Abszess der Orbita und des Stirnlappens bei Empyem von Stirnhöhle und Siebbein, Internat. Centralbl. f. Laryng., Rhin. **32**:294 (Sept.) 1916.

Moreau, E.: Manifestations oculo-orbitaires des sinusites sphénoïdales, Thesis, Lyon, no. 165, 1905.

Mott, F. W., and Robertson, I. M.: Histologic Examination of the Pituitary Gland in One Hundred and Ten Asylum and Hospital Cases, J. Ment. Sc. **69**:296 (July) 1923.

Netter: De la méningite due au pneumocoque (avec ou sans pneumonie), Arch. gén. de méd. **1**:257 and 434, 1887.

Recherches sur les méningites suppurées, France méd. **36**:733 (June 1) 1889.

Neumann, H.: Keilbeinhöhleneiterung und Meningitis, Monatschr. f. Ohrenh. **55**:75, 1921.

O'Malley, J. F., in discussion on Davis, H. J.: Cavernous Sinus Thrombosis Following Left Tonsillitis, Proc. Roy. Soc. Med. (Sect. Laryng.) **6**:176, 1912-1913

Onodi, A.: Die Sehstörungen und Erblindung nasalen Ursprunges, bedingt durch Erkrankungen der hinteren Nebenhöhlen, Arch. f. Laryng. u. Rhin. **17**:260, 1905.

Das Gehirn und die Nebenhöhlen der Nase, Vienna, A. Hölder, 1908, p. 9.

Oppikofer, cited by Fraser (1924).

Orr, D., and Rows, R. G.: Further Observations on Experimental Toxi-Infection of the Central Nervous System, Brain **40**:1 (May) 1917.

Ortmann, P.: Der Diplococcus pneumoniae bei eitriger Meningitis, Virchows Arch. f. path. Anat. **120**:109, 1890.

Palletta, G. B.: Exercitationes pathologicae, Mediolani, 1820, p. 1.

Panas: Phlegmon orbitaire; méningo-encéphalite consecutive; nervite optique avec amaurose; perforation spontanée par osteite des os du crâne, Bull. Soc. de chir. de Paris **2**:507, 1873.

Diagnostic d'une thrombose des veines ophthalmiques et des sinus caverneux, Semaine méd. **5**:255, 1885.

Paunz, M.: Zur Aetiologie der Orbitalphlegmone, Ztschr. f. Augenh. **16**:65, 1906.

Pekosławsky, L.: Caries Ossis sphenoidalis als Ursache von Meningitis Purulenta, Kron. lek. **14**:364, 1893; abstracted, Internat. Centralbl. f. Laryng. **10**: 288, 1894.

Peters, E. A.: Sphenoidal Sinus Empyema in Cerebro-Spinal Meningitis, J. Laryng. & Otol. **35**:11 (Jan.) 1920.

Pickworth, F. A.: Perforation of the Pituitary Fossa by a Septic Lesion of the Sphenoidal Sinus Associated with Recent Epilepsy and Insanity, J. Laryng. & Otol. **43**:186 (March) 1828; reported by Pickworth (1932, case M. L.).

Confusional Insanity with Empyema of the Sphenoidal Sinus, Brit. M. J. **1**: 721 (April 20) 1929.

Sinusitis in Mental Disorder: II. The Pathology of the Nasal Sinuses and Its Relation to Mental Disorder, J. Ment. Sc. **78**:653 (July) 1932.

Organisms in Chronic Bone Infections of the Sphenoid, J. Laryng. & Otol. **50**: 206 (March) 1935.

Pitt, G. N.: An Analysis of Fifty-Seven Fatal Cases of Ear Disease, and of the Complications Which Led to Death, Brit. M. J. **1**:643 (March 22) 1890.

Popham: Sudden and Complete Amaurosis; Scrofulous Tumor Compressing the Optic Nerves; Abscess of the Pituitary Gland; Caries of Body of Sphenoid Bone, Dublin Quart. J. M. Sc. **20**:220, 1855.

Powell, W. E.: A Case of Fatal Meningitis Following Submucous Resection of the Nasal Septum; Post-Mortem Discovery of Latent Sphenoidal Sinusitis, J. Laryng. & Otol. **37**:39 (Jan.) 1922.

Ramdohr, P.: Von den Nasennebenhöhlen ausgehende intracranielle Komplikationen; Sinus cavernosus Thrombose bei akuten Keilbeinhöhlenempyemen; Meningitis der vorderen Schädelgrube mit sensiblen Jacksonanfällen, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **108**:271, 1921.

Rathke, H.: Nachträgliche Bemerkungen zu dem Aufsatze über die Entstehung der Glandula pituitaria, Arch. f. Anat., Physiol., u. wissensch. Med., 1839, p. 227.

Raymond, P.: Carie de sphénoïde; méningite de la base; thrombose des sinus caverneux, exophtalmie double rapide, Bull. Soc. anat. de Paris **60**:227, 1885; cited by de Lapersonne: Phlébite suppurée des veines ophtalmiques et des sinus caverneux, Arch. d'opht. **5**:436, 1885.

Reinhardt, C.: Cancroid des linken Oberkiefers, Virchows Arch. f. path. Anat. **16**:553, 1859.

Ridpath, R. F.: Sphenoid Sinusitis with Peculiar and Unique Symptoms, Tr. Am. Laryng., Rhin. & Otol. Soc. **33**:345, 1927.

Robertson, C. M., in discussion on McBean; in discussion on Pollock, H. L.: Ann. Otol., Rhin. & Laryng. **30**:744 (Sept.) 1921.

Rodger, T. R.: Cavernous Sinus Thrombosis [case 4], J. Laryng. & Otol. **36**: 169 (April) 1921.

In discussion on Colledge, L.: Septic Sinus Thrombosis, Brit. M. J. **2**:460 (Sept. 9) 1922.

Rouge: Periostite du sinus sphénoïdal, Bull. Soc. méd. de la Suisse Rom. **5**: 379, 1871; Union méd., Paris **13**:163 (Feb. 3) 1872.

Roughton, E. W.: Meningitis Secondary to Sphenoidal Sinus Disease, Clin. J. **35**:126, 1909-1910.

Russell, J.: Phlebitis of the Cerebral Sinuses, M. Times & Gaz. **1**:614 (June 8) 1878.

Ruttin, E.: Akute Otitis und Keilbeinhöhleneiterung; Kavernosusthrombose; Meningitis; Exitus, Monatschr. f. Ohrenh. **54**:975, 1920.

Sandford, A. W.: Case of Double Optic Neuritis from Caries of Sphenoidal Cells and Intra-Cranial Abscess, Tr. Ophth. Soc. London **14**:119, 1894.

Schaeffer, J. P.: The Sphenoidal Sinus and the Temporal Lobe, J. A. M. A. **76**:1488 (May 28) 1921.

Schaeffer, M.: Zur Diagnose und Therapie der Erkrankungen der Nebenhöhlen der Nase, mit Ausnahme des Sinus maxillaris, Deutsche med. Wchnschr. **16**: 905 (Oct. 9) 1890.

Ueber acute und chronische Erkrankungen der Keilbeinhöhlen, ibid. **18**:1061 (Nov. 24) 1892.

Schech: Zur Pathologie der Keilbeincaries, München. med. Wchnschr. **45**:843 (July 5) 1898.

Schillinger, R.: Otitic Meningitis, Arch. Otolaryng. **25**:455 (April) 1937.

Schlagenhaufer, F.: Ein Fall von cystischer Entartung der Schleimhaut der Nase und ihrer Nebenhöhlen, Wien. klin. Wchnschr. **12**:867 (Aug. 31) 1899.

Scholz: Caries des Keilbeins, Eitersenkung durch die Fossa sphenomaxillaris, Perforation des Sinus cavernosus, Pyämie und Meningitis; tödtliche Blutung aus dem Sinus cavernosus, Berl. klin. Wchnschr. **9**:516 (Oct. 21) 1872.

Schröder, H.: Erscheinungen von Seiten des Bulbus und der Orbita bei Erkrankungen der Keilbeinhöhlen, Ztschr. f. Ohrenh. **53**:73, 1907.

Schulze, W.: Rapid verlaufende Erkrankungen der Nasennebenhöhlen mit cerebralen Komplikationen [case 1], Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **5**:48, 1912.

Schwabach, (*a*) in discussion on Herzfeld: Ueber Empyem resp. Caries der Keilbeinhöhle [case 2], Berl. klin. Wchnschr. **32**:901 (Oct. 14) 1895.

(*b*) in discussion on Herzfeld: Empyem resp. Caries der Keilbeinhöhle, Internat. Centralbl. f. Laryng., Rhin. **11**:95, 1895.

Shambaugh, G. E., in discussion on McBean.

Sharp, in discussion on Watson-Williams, P.: Latent Sinusitis in Relation to Systemic Infections, Especially Rheumatoid Arthritis, Proc. Roy. Soc. Med. (Sect. Laryng.) **12**:220, 1919.

Sieur and Jacob: Recherches anatomiques, cliniques et opératoires sur les fosses nasales et leurs sinus, Paris, J. Rueff, 1901, p. 299.

——and Rouvillois: Traitement chirurgical des antrites frontales: Etude critique des accidents consecutifs, Ann. d. mal. de l'oreille, du larynx **37**:549, 1911.

Simon, J.: Case of Polypus Nasi, Extending Upwards, and Producing Absorption of the Body of the Sphenoid Bone, and Obliteration of the Internal Carotid Artery, Followed by Abscess in the Brain, Brit. M. J. **1**:471 (June 12) 1858.

Smith, F.: Management of Chronic Sinus Disease, Arch. Otolaryng. **19**:157 (Feb.) 1934.

Basal Meningitis: Some Considerations and a Proposed Management, J. A. M. A. **107**:189 (July 18) 1936.

Stevenson, E., and Adair-Dighton, C. A.: Actinomycotic Meningitis Due to Sphenoid Sinus Suppuration Causing Death Four Days After Excision of the Right Eye, Ophthalmoscope **9**:403 (June) 1911.

Stoerk, K.: Die Erkrankungen der Nase, des Rachens, des Kehlkopfes und der Luftröhre, in Nothnagel, H.: Specielle Pathologie und Therapie, Vienna, A. Hölder, 1897, vol. 13, p. 244.

Stucky, J. A.: Some Mental Symptoms Due to Disease of Nasal Accessory Sinuses [cases 1 and 11], M. Rec. **70**:820 (Nov. 24) 1906.

Meningeal and Cerebral Complications Involving the Silent Area of the Brain (Anterior Frontal Cerebral Lobes) as Result of Ethmoidal, Sphenoidal, and Frontal Sinus Disease; Six Recoveries, Ten Deaths; Result of Eight Autopsies [case 14], Tr. Am. Laryng., Rhin. & Otol. Soc. **17**:402, 1911.

Syme, W. S.: Demonstration eines Präparates von ausgedehnter Meningitis rhinogenen Ursprungs, Internat. Centralbl. f. Laryng., Rhin. **29**:274 (May) 1913.

Thiroloix, J., and Pasquier, C.: Coryza: Suppuration des cellules ethmoïdales et des sinus sphénoidaux; lipémie, Bull. Soc. anat. de Paris **67**:673, 1892.

Thomson, St. C.: Cerebral and Ophthalmic Complications in Sphenoidal Sinusitis: A Contribution Founded on Two Cases with Autopsies, Tr. M. Soc. London **29**:12, 1906; Brit. M. J. **2**:768 (Sept. 29) 1906.

Thost, A.: Ueber seltene Erkrankungen der Keilbeinhöhle, Internat. Centralbl. f. Laryng., Rhin. **29**:181, 1913.

Torrigiani, C. A.: Sindrome ipofisaria da infiammazione delle glandole annesse alle fosse nasali, Arch. ital. di otol. **26**:283, 1915.

Toubert: Contribution à l'étude des complications endocrâniennes de la sinusite sphénoïdale, Arch. gén. de méd. **186**:385, 1900.

Trautmann, G.: Akute Keilbeinhöhleneiterung mit intrakranieller und orbitaler Komplikation, Arch. f. Laryng. u. Rhin. **20**:381, 1908.

Treitel, L.: Ueber Hirnerkrankungen nach Naseneiterungen, Berl. klin. Wchnschr. **33**:1139 (Dec. 21) 1896.

Turner, A. L., and Reynolds, F. E.: (*a*) A Study of the Paths of Infection to the Brain, Meninges and Venous Blood Sinuses, from Neighboring Peripheral Foci of Inflammation: II. Suppuration in the Right Posterior Ethmoidal and Sphenoidal Sinuses; Orbital Abscess; Cavernous Sinus Thrombosis; Acute Leptomeningitis; Operation; Death; Autopsy, with Microscopic Examination of the Ethmoidal and Sphenoidal Air Sinuses, Orbit, Cavernous Blood Sinuses and Meninges, J. Laryng. & Otol. **41**:442 (July) 1926.

(*b*) Furuncle of the Right Nasal Vestibule; Septic Thrombosis of the Cavernous Blood Sinuses: Leptomeningitis; Death; Autopsy, ibid. **41**:73 (Feb.) 1926.

A Study of the Paths of Infection to the Brain, Meninges, and Venous Blood Sinuses from Neighboring Peripheral Foci of Inflammation: IV. Acute Infection of the Left Sphenoidal Air Sinus; Cavernous Sinus Thrombosis with Organization of the Clot; Leptomeningitis; Operation; Death; Autopsy, with Microscopic Examination of the Cavernous Blood Sinuses and Sphenoidal Air Sinuses, ibid. **42**:181 (March) 1927.

(*a*) VI. Meningococcal Leptomeningitis; Death; Autopsy. Inflammatory Changes in the Sphenoidal Air-Sinuses and Early Cavernous Sinus Thrombosis (Streptococcal): A Case Illustrating Double Infection, with Microscopic Examination of the Meninges, Sphenoidal Air Cavities and Cavernous Blood Sinuses, ibid. **43**:34 (Jan.) 1928.

(*b*) VII. Acute Suppuration in the Accessory Sinuses: Cavernous Sinus Thrombosis; Acute Leptomeningitis; Death; Autopsy, with Microscopic Examination of the Ethmoidal and Sphenoidal Air Sinuses and the Cavernous Blood Sinuses, ibid. **43**:565 (Aug.) 1928.

Vail, D. T.: Types of Orbital Abscess and Exophthalmos Due to Intranasal Suppurative Processes [case 1], Laryngoscope **29**:263 (May) 1919.

Vince: Sinusite sphénoïdale avec perforation de l'apophysis basilaire, Clinique, Brussels **14**:156, 1900.

Vlasto, M.: Meningitis of Sphenoidal Sinus Origin, J. Laryng. & Otol. **42**:746 (Nov.) 1927.

Vossius, A.: Beobachtungen über Erkrankungen der Orbita bei entzündlichen Affectionen der Nase und ihrer Nebenhöhlen (Empyem) nebst einem Fall von Mucocele des Stirnbeins und des Siebbeinlabyrinths, Ztschr. f. Augenh. **4**:1, 1900.

Walb: Ueber Erkrankungen der Keilbeinhöhlen, Deutsche med. Wchnschr. **31**: 85 (Jan. 12) 1905.

Watson-Williams, P.: Nasal, Aural, and Other Focal Sepsis as a Cause of Neurasthenia and Insanity, Brit. M. J. **2**:9 (July 4) 1925.

Weichselbaum, A.: Die phlegmonöse Entzündung der Nebenhöhlen der Nase [case 2], M. Jahrb. **11**:227, 1881.

Ueber einige seltenere Complicationen des Typhus exanthematicus in anatomischer und ätiologischer Beziehung [case 8], Allg. Wien. med. Ztg. **28**:226, 1883.

Wertheim, E.: Beiträge zur Pathologie und Klinik der Erkrankungen der Nasennebenhöhlen, Arch. f. Laryng. u. Rhin. **11**:169, 1901.

Westenhoeffer, M.: Pathologische Anatomie und Infectionsweg bei der Genickstarre, Berl. klin. Wchnschr. **42**:637 (June 12) 1905.

Pathologisch-anatomische Ergebnisse der oberschlesischen Genickstarre-Epedemie von 1905, Klin. Jahrb. **15**:657, 1906.

White, L. E.: Disturbance of the Optic Nerve Caused by Malignant Disease of the Sphenoid: Report of Two Cases, Arch. Otolaryng. **6**:361 (Oct.) 1927.

Whitehead, A. L.: A Fatal Case of Necrosis of the Sphenoidal and Posterior Ethmoid Cells; Phlebitis of the Cavernous Sinus; Pyemia, J. Laryng. & Otol. **19**:179 (April) 1904.

Wilson, H.: Multiple Sinus Thrombosis; Cerebellar Abscess and Meningitis Probably Originating from an Osteo-Myelitis of the Sphenoid, Arch. Otol. **31**:443, 1902.

Wright, J.: A Case of Isolated, Unilateral, Latent Empyema of the Sphenoidal Sinuses with Delirium and Mental Symptoms; Operation; Recovery, Ann. Otol., Rhin. & Laryng. **11**:17, 1902.

Wylie, A., and Wingrave, W.: Peritonsillar Abscess Followed by Osteomyelitis, Necrosing Encephalitis and Meningitis, Lancet **1**:178 (Feb. 1) 1919.

Yerger, C. F.: Intracranial Complications of Nasal Accessory Sinus Disease, Illinois M. J. **40**:395 (Nov.) 1921.

The Intracranial Complications of Suppurative Sphenoid Sinus Disease, ibid. **43**:304 (April) 1923.

Young, G.: The Relation of the Optic Nerve to the Sphenoidal and Posterior Ethmoidal Sinuses, Brit. M. J. **2**:1258 (Dec. 30) 1922.

Zörkendörfer, C.: Zur Bacteriologie der Meningitis suppurativa, Prag. med. Wchnschr. **18**:211 (May 3) 1893.

Zuckerkandl, E.: Normale und pathologische Anatomie der Nasenhöhle und ihrer pneumatischen Anhänge, ed. 2, Vienna, W. Braumüller, 1893, vol. 1, p. 345.

Case Reports

AIR EMBOLISM COMPLICATING PHARYNGEAL OPERATION

MEYER J. STEINBERG, M.D., CHICAGO

REPORT OF A CASE

A well built young man 18 years of age underwent an operation on the tonsils and adenoids on March 14, 1936. The usual preoperative medication of morphine and atropine was employed. Ether anesthesia was induced with difficulty. The surgeon stated that he heard loud, sucking sounds emanating from the pharynx during the operation. When the anesthetization was stopped the patient continued to be cyanotic, and the pulse rate and the respiratory rate increased to 132 and 60, respectively. Oxygen was administered intermittently, and caffeine and epinephrine were given hypodermically.

I saw the patient about four hours after the operation. He was semicomatose; there was generalized moderate cyanosis; the temperature was 100.6 F., the pulse rate 106 and the respiratory rate 48; the pupils were dilated but reactive; definite crepitus was palpated along the course of each jugular vein. No essential abnormalities of the lungs were observed. The cardiac dulness was completely replaced by hyperresonance, except for a triangular area from the fourth rib to the fifth interspace along the left sternal border and extending toward the left nipple. Auscultation revealed loud crackling sounds, confined essentially to the area of the right side of the heart, which were synchronous with the cardiac cycle and persisted after breathing was voluntarily interrupted. A diagnosis of air embolism with air hunger was made. The air was contained in the jugular veins and was trapped in the right side of the heart.

Oxygen was administered continuously by nasal catheter. The first night the patient complained of precordial pain. The respiratory and the cardiac rate diminished, and the color improved. Throughout the second day the physical condition remained unchanged. On the third day the patient was comfortable; the area of hyperresonance over the cardiac area was diminished, but the loud crackling was still audible. There was no change in the crepitus over the jugular veins. On the fourth day the administration of oxygen was discontinued. Cardiac dulness was normal on the fifth day, and the crepitus was no longer audible over the heart. At this time it was felt that the patient could be moved so that roentgenograms could be made. The film of the chest revealed nothing abnormal, and that of the neck showed air along both sides.

The patient left the hospital and made an uneventful recovery.

COMMENT

Approximately 200 cc. of air was sucked into the cervical veins during an operation on the tonsils and adenoids. The air was trapped in the right side of the heart for several days and was sufficient in amount to interfere definitely with the normal activity of that side of the heart. Complete recovery occurred within five days. The principal therapy was administration of oxygen.

5349 Winthrop Avenue.

ATYPICAL MASSIVE ABSCESS OF THE FRONTAL LOBE

J. Allan Bertolet, M.D., Philadelphia

The wide variation of symptoms of abscess of the frontal lobe is well recognized. O. Jason Dixon,[1] described the "thoroughness of the brain in concealing an abscess within itself"; Dan McKenzie [2] called attention to the frequently latent signs of abscess in the "silent" frontal area of the brain and to the fact that a usual event is for the abscess to be discovered accidentally at operation or at autopsy.

Infection and trauma have been well recognized as definite etiologic factors by such authoritative otorhinologists as Eagleton,[3] Myers [4] and Skillern and Coates.[5]

The specific symptoms and signs to look for, recently recalled by William Gordon,[6] are: (*a*) initial vague chill, (*b*) headache, (*c*) vomiting, (*d*) general malaise, (*e*) slow pulse, (*f*) subnormal temperature, (*g*) papilledema, (*h*) abnormalities of the cerebrospinal fluid, (*i*) convulsions and (*j*) coma. In addition, as reported by Dan McKenzie,[2] symptoms of slow cerebration, loss of memory and maniacal disturbances may be encountered. Nearly all these cardinal symptoms, with the exception of headache, were absent or at least unrecognizable in the case now under discussion.

From the Otolaryngologic Service of the Hospital of the Protestant Episcopal Church and the Otorhinologic Service of Dr. George M. Coates, the Graduate School of Medicine and the Graduate Hospital of the University of Pennsylvania.

1. Dixon, O. J.: Brain Abscess as the Otologist's Problem, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1930, p. 47; J. A. M. A. **96**:481 (Feb. 14) 1931.

2. McKenzie, D.: Diseases of the Throat, Nose and Ear, ed. 2, St. Louis, C. V. Mosby Company, 1928, pp. 345-346.

3. Eagleton, W. P.: Brain Abscess, New York, The Macmillan Company, 1922, p. 112.

4. Myers, J. L.: Tracts Other Than Otic for Intracranial Infection, Eye, Ear, Nose & Throat Monthly **3**:566 (Nov.) 1924.

5. Skillern, R. H., and Coates, G. M.: An Enormous Frontal Lobe Abscess Following Infection of the Frontal Sinus; Operation; Recovery, Tr. Am. Laryng. A. **52**:48, 1930.

6. Gordon, W.: Frontal Lobe Abscess (Multiple) Secondary to Frontal Sinusitis, with Report of Case, Laryngoscope **44**:717 (Sept.) 1934.

F. A., a white man aged 44, married, was first seen in the outpatient department of the Episcopal Hospital on Sept. 30, 1936. His chief complaint was of facial disfigurement caused by protruding masses on each side of the nasion, which interfered with vision. Only by questioning was a report of the symptom of headache elicited; the headache was intermittent but extremely severe, occurring only when the patient stood erect or when he sat up to read. It was at once relieved by his lying flat on his back. The patient had never previously consulted a physician. He had had no diseases of childhood. His father was living and well at 76 years; his mother died of asthma at the age of 65. His three children were living and well.

Fig. 1.—*A*, exophthalmos of the right eye and prominent protruding masses in the region of nasion. *B*, anterior view of the protrusion.

About four years before seeking treatment he stumbled over a block of wood in his cellar and struck his nose and forehead against a post, losing consciousness for a short time. He noticed some ecchymosis under the right eye the next day, which lasted three days and then cleared up. It was followed a few days later by ecchymosis of the left eye, which also cleared in several days' time. He then felt entirely well until about one year previous to admission, when he noticed a swelling on the right side of the nasion and subsequently on the left as well. Three or four months after the onset of these swellings he observed exophthalmos of the right eye and about two months before admission the onset of headache, first intermittent and later continuous, over the right side of the skull from the

temporal to the occipital area. This headache promptly disappeared if he lay down.

Inspection of the face showed protruding masses on both sides of the nose in the region of the nasion. They were slightly more prominent on the right side. Definite fluctuation was elicited over each protuberance and from one to the other. Examination of the nose revealed deflection of the nasal septum, with impaction of the right middle turbinated bone. There was a slight mucopurulent discharge in the middle meatus on both sides and in the nasopharynx. The antral washings were clear. The tonsils were infected. The ears were normal. There was a large indurated node in the right submaxillary area, which was believed to be due to several infected teeth. The heart, chest, abdomen and extremities were normal. There was a suggestion of a slight Babinski sign on the right.

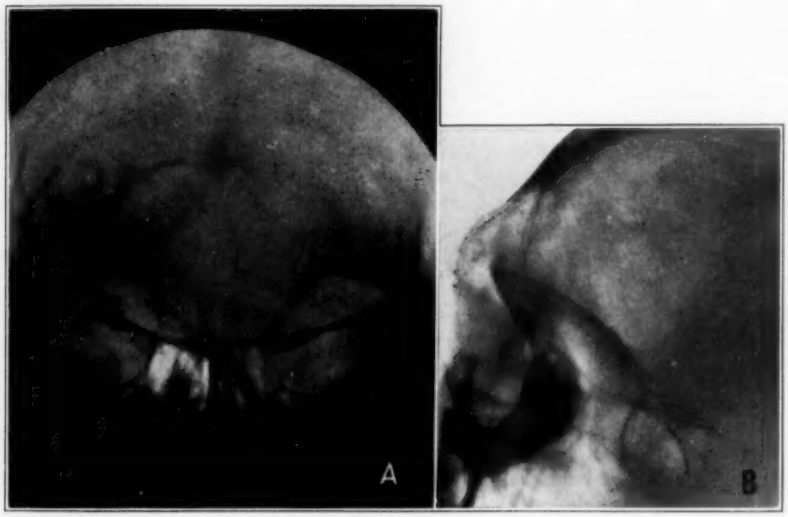


Fig. 2.—*A*, posteroanterior view indicating the extent of erosion of the right orbit and destruction of the frontal septums. *B*, lateral frontal view, indicating destruction of the frontal plates. (Roentgenograms by Dr. M. Lowry Allen.)

Examination of the eyes by Dr. Andrew Knox showed normal visual fields and the absence of refractive error. There was no papillitis or papilledema. Definite exophthalmos of the right eye was noted.

An attempt was made to tap the paranasal protuberance on the right side with a Lichtwitz needle and syringe, without success.

Examinations of the urine and the blood gave normal results. The spinal fluid was clear, the pressure and cell count normal. Wassermann and Kahn tests of the blood gave negative reactions.

The roentgenologic report by Dr. Lowry Allen on October 3 was as follows: "There is marked clouding of both frontal sinuses, together with complete obliteration of the bony detail. In the lateral view there is some moth-eaten appearance of both the anterior and the posterior walls of these sinuses. There are involvement and destruction of the bone of the inner wall of the right orbit.

These findings are indicative of a destructive process involving these sinuses, the exact identity of which is uncertain. The possibilities are: first, a mucocele; second, a low grade infectious process, such as osteomyelitis; third, a tuberculous lesion, and, fourth, syphilis. A new growth at the same time cannot entirely be excluded. Examination of the ethmoid sinuses shows clouding of some of the cells on the right; those on the left are clear. There is cloudiness of the left maxillary sinus, probably due to thickened mucous membrane lining; the right maxillary sinus is clear. The sphenoid sinus is probably normal."

A further report, on October 6, stated: "Additional stereoscopic films fail to cast further light on the nature of the destructive lesion involving the frontal sinuses. The films demonstrate an erosion of the bony walls of both these sinuses, together with destruction of the inner wall of the right orbit.

My chief, Dr. George M. Coates,[7] studied and discussed the roentgenograms with me. He supported my intention to perform a bilateral exploratory operation for possible frontal mucocele. The patient was admitted to the ward October 19 for further study and for the operation. The temperature was normal, the pulse rate 101 and the respiratory rate 18 on admission; on October 20 and on the morning of October 21, the temperature was 98.4 F., the pulse rate 98 and the respiratory rate 16. Unfortunately these data were recorded only twice daily, a serious admission from a diagnostic standpoint, as I now realize. The blood count showed red cells 4,620,000 and white cells 10,960, with hemoglobin, 85 per cent. There were 65 per cent neutrophils, 27 per cent lymphocytes and 3 per cent transitional cells. On October 21, the day of operation, a sample of the blood (taken in error after a large glass of orange juice had been drunk for breakfast) showed a sugar content of 210 mg. per hundred cubic centimeters. The sugar content of the urine was 1.6 per cent. After medical consultation with Dr. John Arnett it was agreed to give 10 units of insulin one hour before operation and to proceed as planned.

Operation.—The patient having been previously prepared and 1/100 grain (0.6 mg.) of atropine with 3 grains (0.19 Gm.) of phenobarbital having been given at 1:30 p. m., gas-ether anesthesia was started at 2:28 p. m. The skin was prepared in the usual manner; the patient was carefully draped. Three cubic centimeters of epinephrine hydrochloride in a dilution of 1:1,000 was injected intracutaneously along the right superciliary ridge; incision was made along the ridge, the blood vessels were ligated and the periosteum was elevated to expose the right side of the frontal area. At this point the frontal mass suddenly extruded through the incision. Respiratory failure occurred immediately, with cyanosis. This was followed almost at once by complete cardiac failure.

Anesthesia was discontinued; carbon dioxide and oxygen were given; one ampule of coramine (a 25 per cent solution of pyridine betacarbonate in diethylamine) was injected; artificial respiration was attempted, and 2 cc. of epinephrine hydrochloride was injected intracardially. The patient was pronounced dead at 3:10 p. m. Exploration was continued after death. The extruding mass was incised, and a great mass of thick, caseohemorrhagic material gushed forth under pressure. There was no evidence of frank pus. Palpation disclosed an enormous cavity. The skull, contrary to deduction from the roentgenograms, was as thin as an eggshell from temporal area to temporal area. The cavity extended posteriorly beyond reach of the index finger but measured 7.5 cm. in depth from the right side of the frontal exposure. As far as could be determined, the entire right side of the frontal lobe and most of the left was either destroyed or compressed beyond palpatory recognition.

7. Coates, G. M.: Personal interview with the author.

The nature of the material is still a matter of doubt. Smears and cultures were sterile. A few epithelial cells of undetermined character were seen. The pathologic findings were: necrotic tissue, nearly all of undetermined histologic character, and much finely granular material. The plug of material taken for histologic examination gave no conclusive evidence.

COMMENT

Death in this case has been ascribed by various colleagues to insulin shock, to too free injection of epinephrine and (the most probable cause) to sudden release of intracranial pressure. The lesion itself has been pronounced by some a massive hematoma and by a group of roentgenologists a broken-down cholesteatoma. Certainly it was not a mucocele. I believe it to have been a sterile abscess of the frontal lobe, the result of trauma, with secondary infection of the nasal sinuses.

The nasal picture of a seemingly mild nonsymptomatic infection was too lightly regarded. Had it been taken more seriously the presence of an abscess must certainly have been suspected. The ignoring of this cardinal etiologic factor in abscess of the brain denied me the satisfaction of a correct diagnosis, though the end result was inevitable.

In conclusion, I repeat the words of Dr. Ralph Butler, quoted by Dr. William Gordon:[6]

> There is no more important phase of rhinology and none worthy of more serious thought and study than that of inflammation of the accessory sinuses and their serious complications. Because of our insufficient knowledge, it is advisable to report our successes and still more so our failures so that evidence may be at hand for the drawing of correct conclusions in regard to the prevention and cure of dangerous complications.

1530 Locust Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

PURULENT OTITIS MEDIA, SINUS THROMBOSIS AND SUPPURATION OF THE PETROUS PYRAMID

ACUTE AND CHRONIC FORMS

SAMUEL J. KOPETZKY, M.D.
NEW YORK

The perusal of the literature for the year under review gives little new material in the way of fundamentals. The literature is replete with clinical material which adds little to the sum of knowledge, and one is puzzled even to find the raison d'être for some of the case reports, since they settle no questions and too often only reiterate accepted facts and customary procedures. In the European field there seems to be absence of intense scientific activity. What little is coming from the university centers is still signed with some well established names. Having continuously studied the foreign literature for years in preparing these surveys, I am struck with the absence of that "docent activity" which formerly was the evident groundwork on which distinguished careers in otolaryngology were built. The Russians are still discussing questions the rest of the occidental scientific world considers settled, and the Japanese are doing considerable experimental work, not well devised, which they undertake without adequate controls and under conditions which would not tend to make the experiment an analogy to what happens when human beings are afflicted with otolaryngologic lesions of pathologic import.

ACUTE PURULENT OTITIS MEDIA

In an endeavor to learn the method and the ways in which an infection from the nasopharynx spreads to the middle ear, Ichikawa[1] inserted an emulsion of virulent Bacillus pyocyaneus into the nostrils

From the Department of Otolaryngology, Beth Israel Hospital.

1. Ichikawa, T.: Experimentelle Studien über den Einfluss des Paraphenylendiamins auf die Nase, Mitt. a. d. med. Akad. zu Kioto **18**:1262, 1936.

of young guinea pigs and then spread the infection to the middle ear by politzerization. The animals were killed after varying intervals of time and examined. He notes that the infection starts at the tympanic opening of the eustachian tube. It then spreads along the anterior and the lower wall (floor) of the middle ear. It then reaches and attacks the membrane at both the fenestra ovalis and the fenestra rotunda and then eventually reaches the antrum and the adjoining spaces. The latter, however, Ichikawa often found free of infection. He also notes a high degree of resistance to infective processes, and he found that the inflamed epithelial cells regenerate quickly. The report is a record of a fine piece of observation, but it hardly throws any light on the question the experimental data sought to clarify. Most infections of the tympanic cavity do not occur in this way. There is usually present a preceding systemic infection from which there are local manifestations in the rhinopharynx. In most instances both the tympanic cavity and the adjacent mastoid cells are hematogenously reached. The old standards set by Eysell, Schwartze and others, based on actual serial microscopic studies, are not negatived by this newer experimental work.

Taking an entirely opposite position and basing their opinions on studies of cultured specimens of bone containing pathologic lesions taken from human beings, Hadjopoulos and Bell [2] report on the mechanics of infection. They feel that the source of infection of the mastoid cells lies in the blood and lymph channels of the adjacent mucosa. They studied the progress of streptococcic infections and could not demonstrate any diffuse dissemination of the streptococci in the tissues. On the contrary, they encountered them strictly limited and sharply localized within definite channels of the tissues. These channels were sometimes venous, often lymphatic and sometimes of indefinite tissue spaces, hard to identify but still sharply and definitely outlined.

From what I have said it is deductible that I tend to agree with the conclusions of Hadjopoulos and Bell on the manner of the spread of infection throughout the aural spaces. The role which the hematogenous route plays in spreading infections is given too little consideration. Only a superficial observer considers that pus bathing the pharyngeal orifices of the eustachian tube is spread grossly and in mass particles into the spaces of the middle ear to cause formation of abscess. It may and it does occasionally occur, but usually the propagation of the infection goes on within the tissues themselves and thus produces the acute lesions of the middle ear and the mastoid.

2. Hadjopoulos, L. G., and Bell, J. W.: Direct Versus Intermediate Pathways in Infections of the Mastoid, Arch. Otolaryng. **25**:601 (June) 1937.

When bacteria reach the tissue of the mastoid cells, they there cause inflammatory reactions.

There is one other factor which needs consideration, based on earlier research studies. Mastoid lesions are rarely primary. The similarity of tissue of the larynx, trachea and bronchial tree, on the one hand, and the pharynx, eustachian tube, tympanic cavity and mastoid cells, on the other, creates a situation wherein a systemic infection by a given organism finds its selected tissue for attack of similar inherent characteristics in both instances; the upper part of the respiratory tract is the first point of manifestation, and the tympanic cavity with its adnexa is the secondary region.

The studies of the infections of the ear during epidemics of influenza are still finding their way into the literature. Steuber [3] presents his study of the otitis which occurred during the epidemic of grip in 1934-1935. He, too, found that the lesion took on a characteristic hemorrhagic form. He reports marked dilatation of the blood vessels, hemorrhage into the middle ear and into the mastoid cells, hemorrhagic blebs on the membrana tympani and at times hemorrhages into the facial canal and into the cochlea. Clinically, the discharge from the middle ear is at first bloody, then serosanguineous and only at the last frankly purulent. At mastoidectomy, Steuber never could demonstrate a coalescence of cells or formation of abscess, nor was periostitis present. He reports that the mortality was no higher than in the other types of otitic infection.

With the expression of opinion as to mortality I cannot quite agree. What Steuber states may be factual observations, but they apply only to the epidemic he reports, and furthermore only provided that this particular type of mastoiditis is clinically recognized by the surgeon and lastly only when surgical intervention is employed promptly. In my own experience during the epidemics from 1918 to 1922, the septic course of the disease often led to fatal outcome because the lesion was unrecognized for what it was. However, Steuber offers another authoritative opinion concerning the hemorrhagic changes typical of the pathologic condition in cases of influenzal otitis and mastoiditis, and his report and observations merit consideration.

Another form of hemorrhagic infection occurs with grip, which runs a less dramatic course. The lesion in the ear has been designated myringitis bullosa haemorrhagica. It consists of a hemorrhagic transudate between the two outer layers of the drum. In a study of 87 cases, Karelitz [4] found that this may be a complication occurring with all types of infections of the upper part of the respiratory tract

3. Steuber: Ueber Grippeotitis, Deutsche mil.-ärztl. Ztschr. **2**:66, 1937.

4. Karelitz, S.: Myringitis Bullosa Haemorrhagica, Am. J. Dis. Child. **53**: 510 (Feb.) 1937.

and that it need not necessarily be a sequela of an actual grippal attack. He cautions against performing paracentesis for such a lesion and advises the therapeutic use of external heat, antipyretics and antineuralgics. During the last winter I saw a few such lesions and am in accord with the outline of therapy which Karelitz advises. In no case did I perform paracentesis, and the lesions promptly cleared and healed. I saw a few other cases in which paracentesis had been done. The clinical course in the latter instances was longer, more stormy and less satisfactory than that in the cases in which paracentesis had not been performed.

Christie [5] cites 5 cases of suppuration of the middle ear which followed an attack of influenza. He believes the lesion in the ear, which he finds often associated with acute maxillary sinusitis, is produced by infection of the maxillary sinus.

The causal relation of acute infections of the middle ear and the intestinal intoxications of infancy is still a subject of wide discussion.

In a symposium on the acute otitis in infantile diarrhea Guillemot [6] states that it is difficult to determine the presence of the otitis as a causative factor and its very diagnosis is difficult. Ribadeau-Dumas [6] feels that after all the other focal infections, such as pyelitis and pulmonary infection, have been ruled out an antrotomy should be performed. This he advocates because it entails no added risk to the patient. Ramadier [6] also holds this view and performs an antrotomy both for diagnostic and for therapeutic reasons if the symptoms do not promptly abate after paracentesis. Bloch [6] and Le Mée [6] report good results from antrotomy. I believe that in this country exception will not be taken to these views. The consensus has been that when a pathologic condition is demonstrable in the spaces of the middle ear antrotomy is indicated, but the use of antrotomy as a diagnostic measure has not yet been accepted as a routine procedure in this type of case. Ten Bokkel Huinink [7] makes a case report which evidences this point of view. He reports 10 cases of purulent otitis media associated with severe diarrhea and other symptoms of malnutrition. In all the cases pus was found in the mastoid process after antrotomy. The organisms involved were mostly of the pneumococcus group. All the patients came to operation when they were moribund, in spite of which 6 were saved. Ten Bokkel Huinink feels that when no cause can be found for the symptoms, and even when myringotomy gives negative results, an antrotomy is indicated. If by operating on

5. Christie, J. F.: The Influenzal Acute Ear, Glasgow M. J. **127**:126, 1937.

6. Guillemot, Ribadeau-Dumas, Ramadier, Bloch and Le Mée: La mastoïdite du nourrisson, Bull. Soc. de pédiat. de Paris **34**:136, 1936.

7. ten Bokkel Huinink, A.: Otitis Media and Toxicosis in Nurslings, Nederl. tijdschr. v. geneesk. **80**:4874, 1936.

patients who come to the surgeon at the point of death 6 of 10 are nevertheless saved, it would seem that significant evidence is thus being adduced to substantiate the point of view of those surgeons who are stressing antrotomy as a therapeutic measure even when equivocal otoscopic findings might impel the belief that the tympanic cavity and its adnexa are normal. Before this topic is left, note is made of a study by György.[8] He studied the pathologic material in a series of cases of empyema of the mastoid antrum in nurslings. He found that the empyema of the antrum extends to the surrounding cells and frequently also acute osteomyelitis is present. This results in intoxication of the entire organism, and surgical therapy is the answer to the problems which the lesion presents. The French literature is rich in studies of the osteomyelitic lesions among very young infants, which will amply repay perusal. The symptomatology, particularly its septic aspects, the mediastinal metastases and other pulmonary reactions and the effects on the gastrointestinal system become increasingly comprehensible as this pathologic phenomenon is better understood.

In 1,555 cases in which operation was performed for mastoiditis, Altmann[9] studied the bacterial causal agent. He found that the causative bacterial flora in the cases fall naturally into groups according to the age of the patients. Thus Streptococcus haemolyticus is the causal agent in 81 per cent of the cases of persons up to the age of 30. This organism accounted for the condition in 24.8 per cent of the cases of persons from that age onward. Of the cases of elderly people, however, Streptococcus mucosus capsulatus (Pneumococcus type III) accounted for the condition in 58.4 per cent. Altmann reports further on the structure of the bone in the patients operated on, saying that one half of the bones evidenced good pneumatization, one third were of a mixed type, with some evident disturbance of pneumatization, and the remainder were of the sclerotic type, varying in degree. Harrell,[10] making a similar study, found that Str. haemolyticus was the cause of acute mastoiditis for most of the patients up to the age of 10 years. It is my own experience that in cases of young subjects —patients ranging in age from infancy almost up to puberty but more noticeably the younger patients—I meet with the pneumococcic type of bacterial flora. For patients in the period from puberty throughout the span of vigorous adult life until well into middle age, I have found the most frequent invading bacterium to be Str. haemolyticus.

---

8. György, E., and von Martyn, R.: Antrum Empyema in Nurslings, Arch. f. Kinderh. **11**:235, 1937.

9. Altmann, F.: Zur Pathologie und Klinik der akuten Mastoiditis, Monatschr. f. Ohrenh. **70**:1465, 1936.

10. Harrell, V.: Hemolytic Streptococcus Mastoiditis, Ann. Otol., Rhin. & Laryng. **46**:194, 1937.

In treating patients in later life, when constitutional diseases have begun to levy their toll and when debilitation is encountered, as in the aged, I again meet the pneumococcic type of infection. There is inherent in the cited observations, as well as in my own experience, a problem the solution of which must be left to the bacteriologists. It involves the transmutation of bacterial forms or types, the influence of the body fluids which constitute their bacterial environment and the other fundamental problems not yet solved.

The differentiation between epitympanic and hypotympanic infections originally was brought to the attention of otologists by Wittmaack. Ciurlo [11] revives interest in this differentiation of the localization of infections of the middle ear. He notes that catarrhal epitympanitis frequently occurs without perforation and without discharge and runs a mild clinical course. Similarly, purulent lesions located in this area often run a mild course due to changes in the mucosa in upper parts of the ear and the disappearance of mastoid purulency which are the results of purely infantile infections. For such lesions, with their mild clinical course, Ciurlo recommends conservative treatment.

Tamari [12] makes an interesting report on a condition which he designates "fibrinous otitis." At the start of every attack of acute otitis, fibrin is present in the mastoid cells. In the usual course of the disease, this fibrin soon disappears; in some instances, however, it may persist and be produced in great quantities, and eventually it appears in the external auditory canal and is deposited as a membrane. This deposit of fibrin acts as a mechanical obstruction to free drainage from the tympanic cavity. Tamari feels that this finding is the result of constitutional factors and debility, and also depends on the type of infecting organism. He studied 32 cases of this condition, in 16 of which epidural or perisinal abscesses were presented. Two of the patients he studied died. All otologists probably have observed such cases without giving this finding the attention its significance merited, and the report of Tamari is stressed to draw attention to this condition.

Mundnich [13] also cites a case report that merits attention. It concerned a 4 year old child with acute purulent otitis media in whom in the third week after the onset of the infection chills and fever developed. The condition was found to be due to malaria. While those in the northern sections of the country only occasonally meet with malaria, in the southern parts not only malaria but filariasis is sometimes concurrent with infections of the middle ear. When otologists

11. Ciurlo, L.: Contributo alla conoscenza delle epitimpaniti acute, Boll. d. mal. d. orecchio, d. gola, d. naso **55**:210, 1937.

12. Tamari, M.: De l'otite fibrineuse, Rev. de laryng. **58**:58, 1937.

13. Mundnich, K.: Akute Mittelohrentzündung und Malaria, Arch. f. Ohrenh. **141**:358, 1936.

in New York are faced with septic fever in the third week of the course of an infection of the middle ear, they look for bacteremia or some other grave condition as the basis of the sepsis. In the South and in the tropical countries, malaria and filariasis are the first factors to be eliminated, and only when these infecting agents are shown to be *absent* is search needed for invasions of the blood stream by the usual pathogenic bacteria. The diagnosis, of course, rests on finding the plasmodium or the filaria in the blood.

A most unusual case report is that of Wamme.[14] After the usual signs of acute purulent otitis media had appeared, a paracentesis was performed, followed by a purulent discharge, which when examined showed a typical intracellular gonococcus. The gonorrheal otitis was successfully treated with an antigonorrheal vaccine, administered parenterally.

From time to time there appear in the literature reports of the curative effect of roentgen rays on acute purulent otitic lesions. The medical profession as a whole has not been impressed with the reports; still the record should be kept. Lucinian[15] treated 31 patients with acute and 8 with subacute lesions. These were all healed without resort to surgical intervention. One treatment was usually sufficient, but in some cases more exposures were required. As a contraindication to therapy, this observer cites high fever, marked leukocytosis and prolapse of the wall of the canal. One wonders whether the patients without high fever, marked leukocytosis or evidence of local periostitis in the form of a droop in the posterior-superior wall of the canal would not have recovered even without the one application of roentgen therapy.

CHRONIC PURULENT OTITIS MEDIA

Evidence substantiating Wittmaack's views is still being recorded. Thienpont[16] conducted an interesting roentgenologic study of the anatomicopathologic picture of the pneumatization of the mastoid process in a series of patients who presented symptoms of chronic progressive deafness.

Lange[17] again writes a dissertation on the development, pathology, manner of growth and prognosis of cholesteatoma. His work is a

14. Wamme, A.: A Case of Acute Gonorrheal Otitis Media, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **28**:195, 1937.

15. Lucinian, J. H.: Treatment of Otitis Media and Mastoiditis by the Roentgen Ray: Analysis of Fifty Consecutive Cases, Am. J. Roentgenol. **36**: 946, 1936.

16. Thienpont, R.: Base anatomo-pathologique de la pneumatisation de la mastoïdite dans l'étude des surdités chroniques progressives, J. belge de radiol. **25**:291, 1936.

17. Lange, W.: Das Cholesteatom des Mittelohrs, Klin. Wchnschr. **15**:1350, 1936.

corroboration of Wittmaack's, and contains much that substantiates the writings of Almour and also my own writings on the questions involved in the study of cholesteatoma. At the Leipzig Ear Clinic, he finds that in a period of time covering ten years 49 per cent of the 73 deaths of patients with chronic purulent otitis media were due to cholesteatoma.

In accordance with all other observers, Lange states that a cholesteatoma can heal of itself if the inert mass is evacuated. He warns that one should never wait for this to happen, however, and that a modified radical mastoidectomy, designed to evacute the epitympanic space—if that is the seat of the lesion—or, when indicated, a tympanomastoidectomy, should be performed in all cases, because a cholesteatoma is always a threat to life.

With this point of view I am fully in accord. To be remembered is the fact that it is impossible to determine, from otoscopic or even from roentgenologic examination or from the combination of both, to what size the cholesteatoma has grown or where it has extended itself. Of course, the removal of a small part of the mass or of a small cholesteatoma may relieve pressure temporarily, and the patient is apt to be lulled into a feeling of false safety because of the abatement of symptoms. The ensuing quiescent period is of varying duration. Eventually symptoms of grave import usually develop. An exactly analogous series of events occurs whether the cholesteatomatous mass is removed by physical means or through the chemical action of carbon tetrachloride or absolute alcohol injected into the epitympanic region in an effort to dissolve the masses.

After a study of 119 cases Blegvad [18] believes that tuberculosis of the spaces of the middle ear can be diagnosed early. He notes as a characteristic otoscopic finding marked swelling of the drum, which is reddish yellow and bulging at the anterior portion of the tympanic membrane. This bulging appears first, to be followed by a similar bulging of the posterior portion of the tympanic membrane. There is marked dilatation of the radial blood vessels.

It is difficult to comprehend the rationale on which the roentgen rays are presumed to act when applied therapeutically to chronic lesions. There are definite pathologic changes which produce the chronicity. The bacterial agent is only one factor. The mutual antagonism of tissues in the encroachment of pavement epithelium on the normal tissues of the middle ear is another factor, and saprophytic secondary infections as well as the fundamental structure of the bone underlying the chronic lesion are all significant. How roentgen rays can restore these abnormal elements to normal interrelations is not clear.

18. Blegvad, N. R.: Remarques sur la tuberculose de l'oreille moyenne, Acta oto-laryng. **24**:187, 1936.

Rabinovitch [19] reports on 280 patients suffering from chronic purulent otitis media who were treated by means of roentgen therapy. The following technic was employed: Four small doses of roentgen radiation given at intervals of ten days are followed by two more doses at intervals of one month each. The results he reports are complete healing in 18.6 per cent of the cases, improvement in 20 per cent and no change in the remaining cases. Of 184 mesotympanic infections, 34 were healed; of 96 epitympanic infections, 18 were healed. After treatment, roentgen examination denoted an increase in the calcium content of the bone. Of 9 patients with cholesteatoma treated, he reports 1 cured. Lucinian,[15] whose report on roentgen treatment of acute attacks has been cited, also reports on 11 cases in which he cured the chronic condition by means of roentgen therapy. The original article should be read by those interested in the details of his technic of application.

Another measure for the treatment of chronic purulent otitis media is reported on by Mertins,[20] namely, the use of urea. This has already been studied by Holder and MacKay, in connection with the treatment of infected wounds. Mertins feels that whatever results are obtainable in cases of chronic purulent otitis media are due more to the thorough removal of the gross and the microscopic débris from the spaces of the middle ear than to the use of the drug. Lewy [21] also reports on its use. He finds that the drug eliminates odor, causes proteolysis of necrotic tissue and stimulates granulations and epithelial growth.

SEPTIC SINUS THROMBOSIS

There seem to be no new aspects of sinus thrombosis to report. Most of the year's literature is concerned with case reports. Woodhall [22] made an anatomic study of the variations to be found in the cranial blood sinuses during the performance of 100 autopsies. He observed a marked difference between the two lateral sinuses in 24 per cent of the specimens he examined. He noted that one or the other lateral sinus was absent in 4 per cent of the cases. In 1 case, in which abscess of the brain and meningitis followed otitis and the Tobey-Ayer test had shown no rise in intraspinal pressure when the left jugular vein was occluded, the autopsy, performed four days postoperatively, showed

19. Rabinovitch, Z. G.: La roentgenthérapie des otites chroniques purulentes, Rev. de laryng. **57**:660, 1936.

20. Mertins, P. S., Jr.: Use of Urea in the Treatment of Chronic Otitis Media, Arch. Otolaryng. **26**:509 (Nov.) 1937.

21. Lewy, R. B.: Use of Urea in the Ear, Nose and Throat, Arch. Otolaryng. **26**:195 (Aug.) 1937.

22. Woodhall, B.: Variations of the Cranial Venous Sinuses in the Region of the Torcular Herophili, Arch. Surg. **33**:297 (Aug.) 1936.

that the longitudinal sinus emptied into the right lateral sinus. Just before the point of junction of these, a small vein went toward the left side, but the left lateral sinus was completely absent. Its place was taken at the jugular foramen by a canal 3 mm. in diameter. Woodhall holds this observation explained the findings from the Tobey-Ayer test.

Woodhall and Seeds [23] believe that roentgen examination can make possible the determination of marked variations in the size of the lateral sinus and that such determinations are important factors when one is interpreting the results of the Tobey-Ayer test.

Natanson [24] studied 4,000 cases of otitis following scarlet fever, the observations covering a ten year period. In 400 of these surgical intervention was eventually employed, and in 10 of the 400 sinus thrombosis developed. He believes that when mastoiditis follows scarlet fever two types of sinus thrombosis may develop. One occurs by the hematogenous route and the other by contiguity. I believe that these two routes to the sinus are followed not only in cases in which sinus thrombosis occurs after scarlet fever, but in all cases. One fact should be stressed: When the vein is infected by the hematogenous route, visual inspection of the exposed wall of the sinus on the operating table gives no sure indication of the presence or absence of a thrombus within the vessel. On the other hand, in cases in which thrombosis is produced by contiguity of infected tissues, a better comprehension of the condition of the vessel's interior is obtainable from the superficial appearance of the sinus.

Tschernjawsky [25] gives an interesting case history. He reports the case of a child suffering from demonstrable septic sinus thrombosis whose middle ear was clinically normal. In an 11 year old girl mastoiditis developed on the right side after scarlet fever; at operation the dura and the sinus were found normal. Four weeks later a typical septic picture developed. During the interval the patient had complained only of occasional pain in the left ear and of headache. Otoscopic examinations of the left ear during this period gave normal findings. At operation on the left ear, a normal mastoid process was found. When the left lateral sinus was uncovered, however, it was found diseased. There was a large thrombus in it, and 10 cc. of pure pus was evacuated. The patient recovered.

---

23. Woodhall, B., and Seeds, A. E.: Cranial Venous Sinuses: Correlation Between Skull Markings and Roentgenograms of the Occipital Bone, Arch. Surg. **33**:867 (Nov.) 1936.

24. Natanson, A. M.: Zur Characteristik der Sinusthrombosen bei Scharlacholitis, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **27**:499, 1936-1937.

25. Tschernjawsky, G. A.: Ein Fall von Sinusthrombose bei klinisch normalem Ohr, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **27**:697. 1936-1937.

There is much that might profitably be discussed regarding this case. In the first place, it adds evidence in support of my belief in the hematogenous route of most otitic infections. After scarlet fever, it is not too uncommon to find purulent foci developing in the kidneys or other organs. I look on a thrombotic lesion involving the left lateral sinus when the mastoid process and tympanic cavity are normal as a metastatic lesion which has accidentally lodged and developed in the left sigmoid sinus while the inciting lesion, with its primary foci of pus, was located on the right side. The finding of 10 cc. of fluid pus in the left sinus reveals that the surgeon reached this lesion late in its development. A thrombus, to have degenerated and to have broken down to form liquid pus, must have been established for some time. It probably had its onset four weeks prior to the appearance of sepsis and at first was to an extent a nonseptic clot. The cure of the condition is not so amazing as the accidental location of the metastatic lesion. In my own experience, when I have first seen a patient late and have found fluid pus within the walls of the vein from a broken-down thrombus, the proximal and distal ends of which seal the system against additional spread of the infection, cure has usually resulted.

Stone and Berger [26] report 5 cases in which a retrograde sinus thrombosis complicated primary thrombosis of the internal jugular vein. Since a thrombus grows both in the direction of and in the direction opposite to the blood stream, the mechanics of the development of such a lesion is comprehended. At the operating table it is often difficult to determine where the diseased section of the vein ends and healthy tissue is in view. Hence a practical lesson may be drawn from this report, and the surgeon when ligating should try to go beyond the point at which the vein appears to be normal.

Michl [27] reports a case of sinus thrombosis in a 13 year old girl, who died of palsy of the abducens and meningitis which developed after jugular resection and thrombectomy. Postmortem examination showed phlegmonous thrombophlebitis in the region of the condylar veins, both the anterior and the posterior being involved. There was also osteomyelitis of the bone around the foramen magnum. In retrospect, the author thinks that perhaps the thrombosis of the condylar veins might have been prevented by radical surgical intervention to the jugular bulb. This I doubt. I know of no surgical procedure which deals adequately with lesions in the tributary veins of the jugular bulb. Even in the usual cases, the weakness of the currently accepted

26. Stone, F. E., and Berger, M. D.: Retrograde Sinus Thrombosis Complicating Primary Thrombosis of the Jugular Vein, Arch. Otolaryng. **24**:141 (Aug.) 1936.

27. Michl, R.: Thrombosis of the Condyloid Veins, Časop. lék. česk. **75**: 1686, 1936.

surgical procedures in the treatment of thrombi which extend into the bulb lies in their inadequacy in the event of one or more of the tributaries of the bulb being involved in the septic pathologic process.

Greenfield,[28] over a period of sixteen years, observed 22 cases of sinus thrombosis, in 19 of which the patients made recovery. He had 17 cases in which the bony structure involved was of a diploic type. In 5 the mastoid process was distinctly pneumatic. This finding accords with the views of most observers. Greenfield stresses the importance of considering the status of the mastoid emissary vein as a factor in continuing otitic sepsis after surgical intervention to remove thrombi from the sigmoid sinus and the jugular vein. He belongs to the school of thought which recommends ligation of the jugular vein in all cases. With this view I cannot differ when considering it from a practical standpoint, since by adopting it the surgeon plays safe in most instances. Nevertheless, I believe that today the cases can be sufficiently differentiated so as to enable a particularization in indications and that in some instances it will be found unnecessary to ligate or resect the internal jugular vein. It would take me too far afield in this review to open this topic more fully. Finally, this observer reports having noted changes in the optic disks in 8 of the 22 cases. Rosenwasser [29] also records that 16 per cent of patients with sinus thrombosis present changes in the optic disk. He is establishing a differential diagnosis between the sepsis due to sinus thrombosis and that which is a symptom of acute bacterial endocarditis. Among the other criteria he cites, in addition to the changes in the optic disk, are the data obtained from using the Ottenberg differential blood culture method. This entails a comparison of cultures of the blood taken from the jugular vein and from the cubital veins. If the number of colonies are equal, it points to endocarditis; if a larger number of colonies are obtained from the blood drawn from the jugular vein and a lesser number are evident in the blood from the arm, it is indicative of sinus thrombosis. There are, of course, other important clinical findings which enter into such a differential diagnosis, but the report of Rosenwasser becomes important in clinical practice when one is faced with a patient with a cardiac disorder who happens to be attacked by suppuration of the middle ear and septic sinus thrombosis as concurrent lesions. The differentiation then becomes important.

Autopsy material is always instructive. Taken apart from the various phases and developments of a clinical study of the course of

28. Greenfield, S. D.: Thrombophlebitis of the Lateral Sinus: Observations in a Series of Twenty-Two Cases, Arch. Otolaryng. **25**:661 (June) 1937.

29. Rosenwasser, H.: Differential Diagnosis Between Thrombosis of the Lateral Sinus and Acute Bacterial Endocarditis, Arch. Otolaryng. **26**:668 (Dec.) 1937.

the disease as the lesion develops and progresses, it is only significantly suggestive, and it helps to furnish means to check on the efficacy of the instituted therapy. In themselves, autopsy reports are but historical records of pathologic changes that have passed in the living body. The autopsy reveals the terminal pathologic picture but does not always clearly show the sequence of events that led to the fatal termination. Such a record of past clinical history is given by Nielsen and Courville,[30] who report on 43 autopsies in cases of intracranial complications of otogenic thrombosis of the lateral sinus. In 15 of these autopsies, evidence of septic meningitis was present. In 10 thrombosis was demonstrable in other venous channels besides the lateral sinus, including the longitudinal, the cavernous and the petrosal sinus. In addition, in 7 cases abscess of the brain was observed. In 2 an intradural abscess was found and in 2 others an epidural abscess was presented. In 4 cases subdural hemorrhage was apparent. In 13 of the autopsies no complications could be seen.

Tobeck [31] reports the case of a woman aged 48, who on the second day after the onset of acute purulent otitis media presented a clinical picture with chills and a swelling over the left mastoid process. At operation the structure of the bone showed severely disturbed pneumatization of the mastoid process, but there was no pathologic change in either the mastoid process or the lateral sinus. Death occurred ten days later. Microscopic examination showed pus filling the cells in the apical portion of the petrous bone and bathing the carotid artery. Thrombosis of the plexus venosus caroticus was demonstrable. At no time were any clinical signs of apicitis present, and Tobeck feels that without the postmortem examination the case would have been designated one of otitic sepsis, without sinus thrombosis. This author emphasizes that in an occasional case apical cells with a meager degree of pneumatization may be the seat of an infection which, spreading adjacently, can cause such a fatal outcome. The cells to which he calls attention are outgrowths in development of pneumatization from the eustachian tube. Here again, I would add, is evidence of the hematogenous spread of infections of the temporal bone. These cells, separated by nonpneumatized bone from the tympanic cavity, the first seat of the infection, present evidence of involvement two days afterward, with chills and other evidence of infection. Furthermore, even had the location of the actual lesion been

30. Nielsen, J. M., and Courville, C. B.: Intracranial Complications of Otogenous Thrombosis of Lateral Sinus, Ann. Otol., Rhin. & Laryng. **46**:13, 1937.

31. Tobeck, A.: Schlagartige otogene Sepsis durch Thrombose im Plexus venosus caroticus im Anschluss an akute Pyramidenzelleiterung, Arch. f. Ohrenh. **141**:177, 1936.

mooted, I doubt that surgical intervention would have availed to save this patient, because even at autopsy the cells in the apex were still not coalesced. Hence roentgen examination would not have been helpful. In other words, diagnostic data failing, no indications were present to suggest surgical intervention to the pyramidal apex.

Engelkes [32] reports 2 cases in which he effected a cure in a novel way. Two patients with sinus thrombosis were cured by parenteral injections of solution of liver extract. Knowing that the agglutination of erythrocytes can be stopped by the administration of liver extract, he employed this method of therapy for what he diagnosed as thrombosis of the superior longitudinal sinus with involvement of the cavernous sinus in addition. He reports 2 cures in such cases and believes that this form of therapy is valuable regardless of whether surgical intervention is or is not employed. I cite this report but question both his diagnosis and the theory on which his therapy is based. It is not the agglutination of erythrocytes that is the factor in the formation of a thrombus. In fact, the erythrocytes do not usually agglutinate. The formation of a thrombus is a defense mechanism of the body to surround and circumscribe an infection in the venous blood channel. Fibrin is thrown out and surrounds the infected area. The erythrocytes are caught in the mesh of fibrin and trapped to form the well known red thrombus. Later, under bacterial activity, this red thrombus breaks down in its center, and organization begins at its periphery. The agglutination of erythrocytes plays no important role in the evolution of this pathologic picture; hence I cannot concede the efficacy of employing an agent to prevent agglutination as a therapeutic measure. If liver extracts actually do cure patients with septic thrombi in the cavernous sinus, as well as surgically reachable blood sinuses, then they must act in a different manner from that on which Engelkes bases his therapy.

SUPPURATIONS OF THE PETROUS PYRAMID

The intense controversy which greeted the earlier reports on suppuration of the petrous pyramid seems to have run its course, and a more objective point of view is beginning to evidence itself in current literature. The opposing groups seemingly are not so far apart as they were. There are still cases reported from autopsy, but in general the surgeons are content to record their factual clinical findings and the technics they used in given cases. Kopetzky [33] presents a critique of the principal surgical procedures used to treat the various

32. Engelkes, H.: Parenterale Leberbehandlung in zwei Fälle von Sinusthrombose, Ztschr. f. Hals-, Nasen- u. Ohren. **41**:348, 1937.

33. Kopetzky, S. J.: Surgical Treatment for Suppurative Petrositis: Critique, Arch. Otolaryng. **26**:294 (Sept.) 1937.

types of petrosal lesions and discusses the indications for the employment of the various ones.

The writers of the case reports found in the year's literature roughly group themselves into two classes: those who diagnose the lesion and operate on it intrapetrosally before rupture has taken place and those who attack it only after rupture has ensued. The latter group still has difficulty in correctly interpreting clinical data and making diagnosis before rupture.

There is now recorded in English, in French and in German comprehensive and fundamental work on which the literature in America and England, France and Europe generally may grow. Thus the work of Kopetzky and Almour [34] in American literature and that of Ramadier [35] in French literature is now supplemented by a fundamental work by O. Mayer [36] in German literature. Mayer presents a complete summation of the subject, with specific indications for the use of surgical intervention. He raises the point and questions why some authorities allude to certain forms of petrositis as being osteomyelitis. He draws attention to the one argument used by these authorities which he calls into question. These observers, he contends, hold that the lesion they describe occurs in bones that are not pneumatized. Mayer's point is, however, that histologic examination of the lesion shows it to differ from true chronic osteomyelitis in that both thrombosis and necrosis are not present.

Mayer brings substantiation to the contention contained in Almour's and my own earlier work that, irrespective of the type of bony structure, both diploic and pneumatized bone at the pyramid will coalesce and will form pus. When free exit for this pus is afforded, often only a granuloma will be found at operation. Such conditions should nevertheless be designated coalescent petrositis. Ramadier limits the designation osteomyelitis to severe conditions occurring in infancy.

Richter,[37] in an examination of tissue from a 66 year old patient who had died of otitic meningitis which followed a mastoidectomy, observed a subcochlear peribulbar empyema which had ruptured into the jugular bulb and there produced thrombosis. In addition, there was present a retrolabyrinthine rupture of pus into the epidural space

34. Kopetzky, S. J., and Almour, R.: The Suppuration of the Petrous Pyramid; Pathology, Symptomatology and Surgical Treatment, Ann. Otol., Rhin. & Laryng. **39**:996, 1930; **40**:157, 396 and 922, 1931.

35. Ramadier, J.: L'ostéite profonde du rocher, Ann. d'oto-laryng., December 1931, p. 1300.

36. Mayer, O.: Ein Beitrag zur Frage der Operation der Pyramidenspitzeneiterungen, Arch. f. Ohrenh. **142**:150, 1936.

37. Richter, H.: Beiträge zur pathologischen Anatomie des Ohres, Arch. f. Ohrenh. **141**:334, 1936.

of the posterior cranial fossa. Pus was found also in the root of the internal auditory meatus. Because no typical mastoiditis was found either at operation or at autopsy, Richter believes that true osteomyelitis, due to infection of the marrow spaces, was present. Here, then, is another authority who believes in the possibility of an osteomyelitic lesion in the pyramid. The absence of mastoiditis needs no additional comment, since primary involvement of the petrosal pyramid is just as likely to occur as primary mastoiditis. Both conditions are rare.

There is another report this year by Mayer,[38] on a petrosal lesion cured after the drainage of a fistula which he found in the tubotympanic region.

Uffenorde[39] divides the cells in the petrous pyramid into four groups, namely, a superior, a posterior and an inferior labyrinthine group and, in addition, a tubal and carotid group. This division is almost analogous to that originally proposed by Almour.[40] Microscopic studies of the development of the bone of the pars petrosa are reported by Collins and Wolff.[41] They found that pneumatization of the apex begins as early as the fourth year, but it is usually more tardy in its development than is the pneumatization of the mastoid process.

Haymann[42] reports on his own material and concludes that meningitis following petrositis is the most frequent endocranial complication of this lesion. Peribulbar abscess or thrombosis of the superior or the inferior petrosal sinus also may occur. Meningitis most often sets in by rupture in the vicinity of the internal auditory meatus. In one of Haymann's cases the pathway of the infection was via the blood vessels of the internal auditory meatus. He observed also that ruptures occur into the floor of the cranial cavity and travel along the vessels and nerves of the jugular foramen, the infection coming from the cells occupying the basal portions of the pyramid. Eagleton[43] discusses the type of petrositis which he terms "osteomyelitis of the inferior surface" of the petrous pyramid. The inferior, sievelike plate of the pharyngeal surface of the pyramid, when eroded by an infection of

38. Mayer, O.: Die Pyramidenzelleneiterungen, Ztschr. f. Hals-, Nasen- u. Ohrenh. **42**:1, 1937.

39. Uffenorde, W.: Die Entzündung in den Zellen des Felsenbeins und ihre Verwicklungen, Hals-, Nasen- u. Ohrenarzt **28**:58, 1937.

40. Almour, R.: Fundamentals of Therapy in Petrositis, Tr. Am. Otol. Soc. **25**:272, 1935.

41. Collins, B., and Wolff, D.: Microscopic Observations of the Developing Petrous Apex, Ann. Otol., Rhin. & Laryng. **46**:444, 1937.

42. Haymann, L.: Ueber Pyramidenzelleneiterungen und ihre endokraniellen Komplikationen, Arch. f. Ohrenh. **142**:304, 1937.

43. Eagleton, W. P.: Osteomyelitis of the Inferior Surface of the Petrous Pyramid, J. A. M. A. **107**:482 (Aug. 15) 1936.

the petrous bone, will permit pus to leak through and form a localized abscess in the roof of the pharynx or in the parapharyngeal space. A lesion located in this area can also cause osteomyelitic caries of the cranial base, or it can produce thrombophlebitis or meningitis. When a parapharyngeal abscess develops, all the localized meningitic signs due to apicitis disappear. In stressing the arguments for a diagnosis of the lesion while it is still intrapetrosal, I[34] have always contended that the sudden disappearance of the characteristic symptoms of apicitis heralds a rupture. Eagleton now substantiates this in the observation just cited. Eagleton reports further that the abscess develops behind the internal pterygoid muscle. The abscess is easily overlooked, and careful search must be made for it. The only comment I desire to add is that I think Eagleton's application of nomenclature is not quite correct. I cannot conceive a pathologic entity which can properly be designated as a surface of bone involved in osteomyelitis. I believe Eagleton's observations are correct; he observes the surface to be eroded by a purulent collection. He actually sees a ruptured pyramid whose surface is affected by the pus impinging on its under surface.

Further discussion of clinical findings is furnished by Uffenorde,[39] who stresses that a disturbance of the fifth nerve can occur from a focus far removed from the gasserian ganglion and that symptoms resulting from this disturbance may be present with any histologic picture. On the other hand, a purulent infiltration of the ganglion, found at autopsy, may during the course of the disease have given no direct symptoms from the fifth nerve.

Pinson [44] reports an unusually interesting autopsy. In a patient who had been treated for a long time by a neurologist for meningitis facial paralysis suddenly developed. This led to an investigation of the ear on that side, involvements of the third, the fifth and the eighth nerve meanwhile developing. The autopsy demonstrated thrombosis of the jugular bulb and suppurative apicitis. Involvement of the third cranial nerve is not encompassed in the clinical picture of suppurative petrositis. Schugt [45] reports a case of Vernet's syndrome. In a patient operated on for mastoiditis there appeared paralysis of the ninth, the tenth and the eleventh nerve. Drainage of a fistula leading into the petrous tip and an epidural abscess resulted in a cure. Fujita [46] noted

44. Pinson, L.: Mastoïdite avec paralysie des nerfs crâniens par thrombose du golfe de la jugulaire et ostéite de la pointe du rocher, Rev. de laryng. **57**:937, 1936.

45. Schugt, H. P.: Glossopharyngeal Neuralgia Associated with Abscess of the Petrous Tip Following Mastoiditis, Arch. Otolaryng. **26**:321 (Sept.) 1937.

46. Fujita, T.: Mucosusotitis with Disturbances of the Glossopharyngeal and Spinal Accessory Nerves, Otologia (Fukuoka) **10**:201, 1937.

that in a 44 year old woman a disturbance of the ninth and the eleventh cranial nerve followed otitic infection. These findings were accompanied by the classic Griesinger sign. Operation revealed a peribulbar abscess, evacuation of which was followed by cure. These reports by Pinson, Schugt and Fujita are important as signifying signs of petrous lesions which pass through an infracochlear route to the apex. I recall the first patient whom Almour and I lost, who gave exactly similar findings during life and whose death started us on our quest for solutions to the problems concerned with suppurations of the petrous pyramid. Barth [47] reports a case of severe pain in the tract of the fifth nerve, paralysis of the abducens and facial palsy. Mastoidectomy, having been done, was revised. The author suspected that he was dealing with an abscess of the brain. He treated the patient, performing a Ramadier operation, and effected a cure. Grabscheid [48] reports a case of tuberculosis of the petrous apex in a patient aged 37.

A different type of case is reported by Traina,[49] in which palsy of the sixth nerve developed fifteen days after mastoidectomy in a 13 year old girl. Radical mastoidectomy was performed without affording relief. Subsequently a revision of the wound was undertaken, and pus was found coming from the area between the cochlea and the carotid artery. This fistulous opening was enlarged, and a retained abscess was evacuated. The palsy of the sixth nerve lasted sixty-five days longer. At no time in the entire history of the case was there any headache or other pain.

Had no palsy of the sixth nerve been present, in the absence of all other characteristic symptoms of petrositis, this fistula would have drained into the tympanic cavity and the pus found egress into the external auditory canal, and there would have resulted a condition which is termed chronic otitis media suppuration. Actually from the pathologic standpoint there would have been a chronic petrosal suppuration emptying through the perforation in the tympanic membrane. I therefore consider this an arrested chronic petrosal suppuration. Only the palsy of the sixth nerve induced the persistent search for the focal lesion which Traina undertook, and thus he could report a recovery. This case again illustrates the fact to which I have called attention before, namely, that every chronically discharging ear in which otorrhea has its onset concurrently with acute mastoiditis and

47. Barth, H.: Akute Otitis media mit Beteilung der Pyramidenspitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:95, 1936.

48. Grabscheid, E.: Tuberculosis of the Petrous Apex: Report of a Case, Arch. Otolaryng. **25**:11 (Jan.) 1937.

49. Traina, S.: Petrosite con vasta zont di rarefazione dell'apice, paralisi dell'abducente, assenza di sintomatologia dolorifica, ipersonnia recidivante, intervento, guarigione, Riv. oto-neuro-oftal. **13**:449, 1936.

which at operation is found to have a pneumatic structure of the bone should be examined specifically for a chronic lesion originating in the petrosal pyramid and palsy of the sixth nerve need not be in the clinical picture to make diagnosis possible. Baylor,[50] discussing at length the symptomatology and bacteriology of petrositis, states that palsy of the abducens nerve is not needed to make a diagnosis of the lesion. Franchini and Riccitelli [51] conclude that if palsy of the abducens nerve appears during the first fifteen days of otitis when no surgical intervention has been performed the patient will get well. They cite a case in point. I have stressed a differential diagnostic point, to wit, that the palsy of the abducens appearing early, even when a mastoidectomy has been performed, is not so much a diagnostic factor of petrositis, while the paralysis of the abducens which appears late in the course of the disease is more apt to be significant. The basis of Franchini and Riccitelli's conclusions is not clear to me.

Arnold,[52] in a study of 17 cases of apicitis, found the fifth nerve gave the symptom of pain in 14 cases. In 5 of these there was evidence that the pain was distributed along the upper and the lower teeth. It should be remarked here again, to stress the point, that in my opinion pain referable to the trifacial nerve suggests only a suspicion of petrosal involvement. It is not characteristically significant, for there are definite cases in which the history discloses no such pain. Of Arnold's patients, 10 died. He adds a note that the abducens nerve is less often involved in petrositis than is the fifth nerve.

Among reports on cases in which rupture has occurred and the pus has spread externally beyond the bony boundaries of the petrous pyramid, the following are instructive. In all of them there is evidence of an earnest endeavor to treat the lesion surgically, but apparently there is difficulty in recognizing the lesion prior to its rupture extrapetrosally. These cases of ruptured lesions spilling over into adjacent tissues are frankly and honestly reported. In analogy it is as if otologic surgeons should await the formation of an extradural, perisinal or subperiosteal extravasation of pus before undertaking operation on a purulent mastoid.

Yamashita and Masayoshi [53] report a case of petrositis accompanied by a retropharyngeal abscess in a woman aged 42. After purulent

50. Baylor, J. W.: Suppuration of the Petrous Pyramid of the Temporal Bone, Internat. Clin. **46**:290, 1936.

51. Franchini, Y., and Riccitelli, E.: Consideraciones sobie un caso de sindrome de Gradenígo, curado sin intervención, quirúrgica, Semana méd. **1**:96, 1936.

52. Arnold, F.: Ueber die Trigeminusneuralgie insonderheit die Zahnschmerzen bei Felsenbeinspitzenempyemen, Dissert., Erlangen, 1936.

53. Yamashita, K., and Masayoshi, I.: Suppurative Petrositis and Otogenic Retropharyngeal Abscess, Otologia (Fukuoka) **10**:110, 1937.

otitis media on the right and mastoiditis, caused by the Streptococcus mucosus (Pneumococcus type III), the chief symptom was a continuous headache. No other signs of petrositis were present. Twenty-nine days postoperatively they noted a swelling in the pharynx appearing as a red, fluctuating mass. This was incised and released pus, which when cultured showed Str. mucosus. By means of a probe inserted through the opening thus created a communication between the pharyngeal abscess and the petrous apex could be demonstrated, and its presence was corroborated by roentgen examination. Cure followed. Bombelli [54] had a case of petrositis. He performed a radical mastoidectomy and found and enlarged a paratubal fistula. There followed a typical infiltration of pus in the neck, which necessitated evacuation through an incision behind the sternocleidomastoid muscle and dissection of the lateral cervical muscles. Cure followed. Monteiro [55] cites 2 typical cases of petrositis in which cure was effected by the extrapetrosal approach of Streit, while Kleinfeld [56] reports recovery in a case of petrositis. The patient, 9 years of age, had a typical history. Recovery followed petrosal drainage of an abscess in the middle cranial fossa, localized over the apical portion of the pyramid. Myerson and Rubin [57] report 5 cures in 6 cases comprising the material of their report. The 5 cures were obtained by extrapetrosal surgical intervention, except in 1 case, in which a radical mastoidectomy revealed a fistula at the tubotympanic orifice, which was successfully drained. In 2 cases an intracranial approach was made, and a subdural trough located a supracochlear lesion; in another case also a subdural approach, by a technic which Myerson has described, was successful, and in another case a high retropharyngeal abscess evacuated the drainage from a lesion of the petrous bone, and the lesion healed. The other patient died.

The rest of the year's literature on petrositis which is deemed noteworthy deals with methods of surgical therapy. Thus Dearmin [58] presents what he calls the logical approach to the cells of the tip of the petrous pyramid, which he had worked out on specimens. After simple mastoidectomy, the lateral sinus is exposed and followed until

54. Bombelli, U.: Un caso di petrosite con fistolizzazione in faringe e infiltrazione latero-cervicale profunda, Ann. di laryng., otol. **36**:56, 1936.

55. Monteiro, A.: Symptomenkomplex der Gradenigo: Streitsche Operation, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **28**:434, 1937.

56. Kleinfeld, L.: Petrositis with Operation and Recovery, Arch. Otolaryng. **24**:503 (Oct.) 1936.

57. Myerson, M. C., and Rubin, H. W.: Further Experiences with Suppuration of the Petrous Pyramid, Arch. Otolaryng. **25**:525 (May) 1937.

58. Dearmin, R. M.: A Logical Surgical Approach to the Tip Cells of the Petrous Pyramid, Arch. Otolaryng. **26**:314 (Sept.) 1937.

the jugular bulb is reached. The entrance to the cells of the tip is then made by directing a 1.5 mm. burr at an angle of 10 degrees anteriorly and 5 superiorly, so that it passes between the bulb and the internal auditory meatus. One may remark that this approach lays the blood vessels of the bulb open to considerable danger. Furthermore, the term "logical approach" can hardly be applied to a surgical procedure which at best would adequately deal with only one known set of lesions. It certainly is obvious it would not be applicable to all. Kramm [59] also has an excellent paper in which he discusses all manners of surgical approach, giving specific indications for the various procedures. Alonso and Regules,[60] while reporting on 5 patients whom they cured, give typical case histories. The merit of this contribution lies in the fact that these authors tried different procedures and endeavored to fit each case with a specific procedure. They employed the procedure of Kopetzky and Almour, that of Ramadier and that of Eagleton. I regret that they made no note comparing the procedures and evaluating each to determine the advantages of one over the other in given circumstances. Langenbeck [61] advocates the intrapetrosal approach. He prefers a combination of the Ramadier and the Almour technic, except that he does not approve the use of the burr as Almour does. He warns that the subdural approach is not without hazard, because of adhesions between dura and bone, which must perforce be torn. This he believes can add an element to spread the infection intracranially. He is in accord with Almour as to the point of maximum drainage, namely, the tubotympanic area, as the point of selection for opening into the region of the tip of the pyramid. Ninger and Hybasek [62] also favor the intrapetrosal approach for suppurations of the petrosal apex. They cite 6 cases. Two of the patients had lesions located in the anterior perilabyrinthine area and 4 in the postlabyrinthine region. Of their 6 patients 5 recovered. They bring a new term into use. It is a happy suggestion. They feel that for anterior labyrinthine petrositis the "minimal operation" is a radical mastoidectomy, while for posterior petrositis the "minimal operation" is a simple

59. Kramm, H.: Die Ausräumung der Pyramidenspitze, Hals-, Nasen- u. Ohrenarzt **28**:73, 1937.

60. Alonso, J. M., and Regules, P.: Les pétrosites, Rev. de laryng. **57**:898, 1936.

61. Langenbeck, B.: Zur operativen Behandlung tiefgelegener Herde in der Pyramidenspitze, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:369, 1937.

62. Ninger, F., and Hybasek, J.: Les pétrosites, Rev. de laryng. **58**:1, 1937; Otogene Felsenbeinentzündung, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:155, 1937-1938.

mastoidectomy. Lempert,[63] whose technic was commented on last year, because it had a prior publication to the one here cited, describes again a technic for complete apicectomy. Lempert's main contention is that all prior procedures are in reality efforts to establish drainage, while his procedure eviscerates the interior of the petrous pyramid, particularly its apicocarotid portion, as completely as one does the interior of a diseased mastoid process. I have employed this operation, as I reported last year, and know that the surgical procedure is feasible, and in my opinion it is necessary particularly when lesions threaten rupture through the anterocochlear region into the middle cranial fossa, anteriorly to the internal auditory meatus.

In view of the large number of my own cured patients, as well as those of other observers whose reports I have studied, all of whom were cured by the institution of adequate drainage, I consider that, while this operation has its definite place, it is not indicated in all cases of suppuration of the apicocarotid region of the pyramid. I further hold that drainage should always be tried first. This failing, the question of employing the Lempert technic comes into consideration.

Myerson,[64] in another communication, states that the operation of Lempert and Ramadier results in an interference with the function of the temporomandibular joint. With this contention I am not in accord. I have performed the operation, and no such result was ever apparent. Of course, when taking down the anterior bony external wall of the auditory canal one must exercise care to confine the procedure to the region below (inferior to) the petrosquamous suture. The temporomandibular joint is above this landmark. Thus trauma to the joint is avoided. Myerson makes the further criticism that by undue exposure of the carotid artery one may expose it to the formation of an aneurysm, because the surgical procedure takes away from the vessel its bony support. That is a misapprehension, because only one side of the artery has its bony support removed. The carotid canal becomes a trough, in which the artery continues to rest. It is the landmark from which bony evisceration proceeds. Finally, while aneurysms have been known to occur when the artery is stripped of its outer coat, this happens because its sympathetic nerve supply is then lost. This is not the case when either a Ramadier or a Lempert operation is properly done. Hemorrhage has been reported, but in my reports and in those of Ramadier and of Lempert it has not been encountered.

---

63. Lempert, J.: Complete Apicectomy (Mastoidotympanoapicectomy), New Technic for Complete Exenteration of the Apical Carotid Portion of the Petrous Pyramid, Arch. Otolaryng. **25**:144 (Feb.) 1937.

64. Myerson, M. C.: Suppuration of the Petrous Pyramid: Some Views on Its Surgical Management, Arch. Otolaryng. **26**:42 (July) 1937.

Finally Guild [65] makes a histologic study of the region between the cochlea and the carotid artery and calls attention to the close proximity of these two structures. He warns against surgical intervention between the artery and its adjacent cochlear capsule. While the histologic analysis is correct, the factual observations of the surgeon place the point of attack on the artery at some distance away from the region demonstrated by Guild. It is possible that in an occasional instance there will be no available space between the structures to which he draws attention, but in the majority of cases the surgeon who follows either the technic of Ramadier or that of Lempert need not fear this danger. In view of the large number of operations involving the carotid artery in this area, I consider the objections raised by Guild more theoretic than practical.

65. Guild, S.: Hitherto Unrecognized Danger in the Operation of Ramadier for Suppuration of the Petrosal Pyramid, Acta oto-laryng. **25**:561, 1937.

71 East Eightieth Street.

News and Comment

CASSELBERRY PRIZE FUND

The sum of $500 has accrued from the Casselberry Fund, for encouraging advancement in the art and science of laryngology and rhinology, and is now available, in part or as a whole, for a prize award or decoration or to defray the expense of original investigation or research in the domains mentioned. Theses or reports of work done must be in the hands of the secretary, Dr. James A. Babbitt, 1912 Spruce Street, Philadelphia, before February 1 of any given year. Any further information desired will be gladly furnished by the secretary.

Abstracts from Current Literature

Ear

DEVELOPMENT OF HEARING IN OPOSSUM. EDWARD MCCRADY JR., ERNEST GLEN WEVER and CHARLES W. BRAY, J. Exper. Zoöl. **75**:503 (April) 1937.

The development of auditory function was studied in pouch young opossums by observation of the reflex motor responses to pure tones produced by a loud speaker and of the electrical responses recorded from the round window or the acoustic tracts during stimulation with sound. Both types of test indicated an early appearance of sensitivity to tones of the middle range and progressive growth of sensitivity to higher and lower tones. The complete range was attained at weaning, when the young left the pouch. The results support the view that tones of from 1,000 to 3,000 cycles are focalized in the distal portion of the basal coil and that the range is extended in both directions by differentiation of neighboring structures. The foci for all tones of from 3,000 to 25,000 cycles are crowded into the proximal portion of the basal turn of the cochlea, and the foci for lower tones are spread out toward the apex. The sensitivity of the opossum's ear is less than that of the guinea pig's, especially for low tones. The lower limit for the opossum is about 100 cycles. Maximum sensitivity is at first near the middle of the range and with increasing age shifts upward about two octaves (to about 7,000 cycles). At all ages and at all frequencies of stimulation there is a nearly linear relation of the electrical response to the intensity of the stimulus. This indicates a highly efficient electroacoustic apparatus.

WYMAN, Boston. [ARCH. NEUROL. & PSYCHIAT.]

PATHOLOGIC CHANGES IN THE EAR IN LATE CONGENITAL SYPHILIS. O. MAYER and J. S. FRASER, J. Laryng. & Otol. **51**:755 (Dec.) 1936.

Mayer and Fraser show that the principal changes in the ear due to late congenital syphilis are osseous lesions, namely, osteomyelitis gummosa, periostitis gummosa and periostitis productiva. Other changes are usually caused by trouble in the bone which surrounds cavities and canals, invasion of which is easy. Thus the authors found serous labyrinthitis, either florid or cured, in the cochlea and vestibule in all patients examined. This was probably due to the setting up by miliary gummas in the labyrinthine endosteum of a serous exudation leading to slight proliferation of tissue in the scalae, ectasia of the ductus cochlearis and, lastly, pronounced shrinking of the membranes, atrophy of the organ of Corti and degeneration of the maculas. In the semicircular canals the authors found chiefly productive inflammation developing in the periosteal layers. Constant endosteal proliferation of tissue and bone leads to concentric narrowing of the lumen. The endolymphatic space usually remains patent, and the endolymph flows freely. More rarely, a gummatous inflammation, consisting of miliary gummas or large ones which fill the entire canal, accompanies this periostitis productiva. This syphilitic tissue may erode the semicircular canal on the interior, thus causing eruptions into neighboring marrow spaces. Gummatous osteomyelitis leads to extensive changes of structure in the capsule of the labyrinth. The process attacks, preferably, the region of the semicircular canals at the margin of the periosteal capsule but invades the labyrinth. Deficient replacement of the resorbed bone may lead to osteoporosis of all the bones of the ear and of the petrous portion of the temporal bone. In the ossicle osteomyelitis generally causes ankylosis of the malleoincudal joint. In the oval window the authors found formation of osteophytes.

There was no stapedial ankylosis, but specific infiltration of the annular ligament may have caused softening. In the os tympanicum there were gummas, necrosis of the bone and consequent cholesteatoma in the external meatus. The nervous system showed lesions due to miliary gummas in the ganglion spirale and to gummatous inflammation in Rosenthal's canal and the internal meatal fundus, particularly in the central and the superior fovea. The disorder is extremely insidious. In all cases observed the authors met fresh inflammatory processes, in spite of the duration of the disease; particularly, subendosteal periostitis in the semicircular canals seems to flare up again and again. Fresh lesions are to be seen close to healed ones. Lesions may be caused anywhere while others nearby may subside. This explains the lack of uniformity of the clinical results, particularly in those revealed by functional examination of the labyrinth.

EDITORS' ABSTRACT.

OTOMICROSCOPY IN THE LIVING. E. LÜSCHER, J. Laryng. & Otol. **51**:779 (Dec.) 1936.

Lüscher applies the name otomicroscopy to the inspection of the ear of the living subject with magnification of from 10 to 50 times. Owing to the great progress of microscopy with supercadent light it was possible to construct a simple ear microscope which can be used for clinical purposes and in routine practice. The handling of the new ear microscope is no more difficult than the use of the ordinary otoscope and is done in the same way. In spite of the fact that otomicroscopy is still in its infancy, considerable progress has been made, and many differential diagnostic difficulties have been removed in a simple way. The author describes the otomicroscopic picture of the tympanic membrane as it appears normally and in some pathologic conditions, such as acute otitis media, mastoiditis and tuberculosis of the tympanic membrane.

EDITOR'S ABSTRACT.

MASTOIDITIS IN INFANTS. RIBADEAU-DUMAS, RAMADIER, GUILLON and L. MELLETIER, Bull. Soc. de pédiat. de Paris **34**:33 (Jan.) 1936.

A series of 109 cases of mastoiditis in infants is reported. Fourteen were discovered at autopsy; in 3 of these there had been otitis media, and in 11, no signs of otitis media. Sixty-five infants were operated on; 38 of these recovered and 27 died. Of the latter, 18 had bronchopneumonia, 2 had meningitis and 7 had diarrhea. Of the 38 who were cured, only 6 had bronchopneumonia and 5 diarrhea. Ten per cent of those operated on had latent mastoiditis; they were submitted to antrotomy because of diarrhea with severe dehydration and acidosis for which no cause had been found and which did not improve after a few days of the usual treatment. Only 2 mastoid antrums which were operated on showed no signs of involvement, and in 1 of these there was a free flow of pus forty-eight hours after operation. In 10 cases in which cultures were obtained, there were pneumococci in 4 and streptococci in 6. Of the infant mortality in the hospital 11.5 per cent was assigned to mastoiditis. The antrotomies were done under local anesthesia.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

CONCERNING MASTOIDITIS IN INFANTS. WEILL-HALLÉ, PAPAIOANNOU and MOUCHOTTE, Bull. Soc. de pédiat. de Paris **34**:122 (Feb.) 1936.

Figures are cited to show that mastoiditis is relatively rare in infants. In 1935, of 796 children in a crèche, 66 had suppurative otitis media and 12 mastoiditis, and in 1934, of 816, there were 105 with suppurative otitis media and 12 with mastoiditis. While rhinopharyngeal infection with or without otitis media and mastoiditis may be directly related to digestive disturbances in infants, there

does not appear to be primary mastoid infection in such cases. Antrotomy is recommended only when definite physical signs indicate its necessity. More harm than good may result from indiscriminate operation on the mastoid when there are no definite local findings to warrant the procedure.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

MEDICAL TREATMENT OF MASTOIDITIS. LÉON TIXIER, Bull. Soc. de pédiat. de Paris **34**:493 (Oct.) 1936.

The discrepancy between the many mastoid operations done by some physicians and the few performed by others is explained by the fact that the latter temporize and obtain good results without operation in many cases while the former resort to surgical intervention early, without definite indications of its necessity. Mastoiditis without otitis media is certainly not frequent, and the necessity for mastoid operation when no physical signs are present is rare.

Among the infants covered by this report, of all who had definite mastoiditis none had intestinal symptoms. As 90 per cent of mastoiditis is believed to be due to streptococci, vaccine therapy was used, and good results are claimed for it; recently the administration of sulfanilamide in addition to the accepted treatment has been beneficial. Instances are mentioned in which after operation was recommended the child was spared such a procedure because of watchful waiting and medical treatment. The author recalls many winter seasons in his city practice in which a considerable number of children with suppurative otitis media were treated and not a single mastoid operation was performed.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

DIPHTHERITIC MENINGITIS OF OTITIC ORIGIN AND OTHER COMPLICATIONS OF DIPHTHERITIC OTITIS. V. HORNÍČEK and V. JEDLIČKA, Časop. lék. česk. **74**: 1186 (Nov. 8) 1935.

Scarlet fever decreases resistance to infection, and the scarlet fever-diphtheria sequence causes infection of the middle ear in 7.2 per cent of cases of this disease, while uncomplicated diphtheria is incriminated in 5 per cent of cases, according to most authors. The most toxic effects are noted in patients in whom diphtheria appears at the beginning or during the first week of scarlet fever. When diphtheria comes on during desquamation the effects are often milder. The authors observed 3 cases of severe complications in the middle ear in children with scarlet fever and diphtheria combined: (1) otitis and mastoiditis purulenta, pachymeningitis, leptomeningitis circumscripta purulenta and meningoencephalitis (diphtheria organisms being recovered culturally after autopsy); (2) bilateral purulent otitis and mastoiditis and endocarditis verrucosa mitralis, from which pure cultures of diphtheria organisms were recovered; (3) ethmoiditis and mastoiditis, with a postoperative orbital sinus, from which the patient recovered.

STULIK, Chicago. [AM. J. DIS. CHILD.]

OTITIC COMPLICATIONS OF SCARLET FEVER. F. NAVRÁTIL, Časop. lék. česk. **74**: 1226 (Nov. 8) 1935.

After postscarlatinal neuritis the next most common otitic complication of scarlet fever is otitis media. During 1934 the number of cases of otitis media was 233, or 12.4 per cent, in a series of 1,980 cases of scarlet fever. In 3 of these the condition was catarrhal and purulent; in 36, catarrhal, and in 192, suppurative. Of the cases in which it was suppurative, in 106 it was early and in 88 late. Thirty paracenteses were done, with 2 deaths in the series.

STULIK, Chicago. [AM. J. DIS. CHILD.]

SURGICAL TREATMENT OF SUPPURATION IN PYRAMIDAL APEX. O. MAYER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:150, 1936.

Mayer reports the clinical history of a boy aged 16 who had a large abscess at the pyramidal apex and who presented the typical Gradenigo syndrome. The apical abscess was opened by introducing a sharp curet along a fistular passage into the eustachian tube and proceeding laterally around the exposed carotid into the cavity of the abscess. Complete cure was effected. The case is noteworthy because of (1) the slight pneumatization, (2) the manner of detection of the abscess and (3) the manner of exposure. Regarding the second point, the author says that in searching for deep foci of pus attention should be given to the appearance of small dots of pus, for these help in locating fistulas, and their probing leads to the discovery of the focus of the pus. EDITOR'S ABSTRACT.

VESTIBULAR DISORDERS IN HEREDITARY DEAFNESS. E. MÜLLER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:156, 1936.

Müller points out that studies of other investigators revealed that vestibular disturbances are more frequent in acquired than in congenital deafness. His own investigations, however, revealed that the concurrence of hereditary deafness and disturbances in the apparatus of equilibrium is not at all rare. He describes two unusual types of hereditary vestibular lesion, one of which he observed in a family with hereditary degenerative deafness and the other in a family with sporadic deafness. In the first family he observed in 4 deaf members of one generation a vertical nystagmus that was dependent on the position of the body. The children of both families showed a considerable improvement of the hearing capacity. However, the functional impairments of the vestibular apparatus recurred in slightly changed form in the descendants. EDITOR'S ABSTRACT.

TUBERCULOSIS AND EAR. E. URBANTSCHITSCH, Monatschr. f. Ohrenh. **70**:1287 (Nov.) 1936.

Urbantschitsch first takes up lupus of the lobule of the ear, stating that between 1907 and 1913 he observed 4 cases; in each he employed surgical methods and Finsen irradiation. He says that in recent years the disorder has disappeared, probably because earrings are no longer worn and treatment is begun earlier. In cases of histologically demonstrated tuberculosis of the mastoid process, he noticed a considerable discrepancy between the subjective and the objective course of the disorder. He found a favorable general condition in the presence of great destruction. He observed "clinically primary" tuberculosis of the middle ear in 2 nurslings and in a woman aged 70. The prognosis in that disease seems favorable. The author reports 3 cases of miliary tuberculosis in which the dissemination took place after the surgical intervention. The condition terminated fatally, but the process in the ear was such that the operation could not have been avoided without endangering the life of the patient. The author describes nontuberculous mastoiditis in a case of tuberculosis. This report demonstrates that in cases of advanced general tuberculosis otitis media does not necessarily have to be tuberculous. Finally the author cites a family history which indicates that deaf-mutism may develop on a tuberculous basis. EDITOR'S ABSTRACT.

IMPROVEMENT OF FACIAL PARALYSIS BY SURGICAL INTERVENTION. I. T. DOROSCHENKO, Monatschr. f. Ohrenh. **70**:1303 (Nov.) 1936.

Doroschenko directs attention to the fact that surgical intervention on the ear occasionally results in paralysis of the facial nerve. If this does not disappear after a time and if conservative measures fail, the author thinks surgical measures should be resorted to. He reviews the different surgical methods that have been recommended and then reports his own observations in 14 cases of chronic facial

paralysis. The paralysis had existed for from six months to eight years. Conservative measures in the form of massage, electrotherapy, medication and so on were always tried first. When these failed, the author resorted to neurotization according to Rosenthal's method, to autoplasty according to the method of Payr, Kirschner, Stein and Burgh or to radical trepanation of the ear with exposure of the canal of the facial nerve and liberation of the nerve from pathologic processes. The author demonstrates the favorable results of the surgical treatment by reviewing several cases.

EDITOR'S ABSTRACT.

PATHOLOGIC STRUCTURE OF TUMORS OF EXTERNAL AUDITORY MEATUS. K. LÖWY, Monatschr. f. Ohrenh. **70**:1319 (Nov.) 1936.

Löwy reports 4 cases of rare types of tumor of the external auditory meatus which recently came up for observation at his clinic. The first patient had a fibroma which grew again after removal. Histologic examination revealed no sign of malignancy. After reviewing the literature on such tumors, the author reports 2 cases in which a typical nevus was found in the external auditory meatus. He says that nevus of the external auditory meatus is extremely rare. To be sure, since the nomenclature and the origin are still somewhat obscure, it is possible that such tumors have been reported under different names. In the fourth case the tumor was a fibroma. This case somewhat resembled the first one in that a tendency to relapse existed. However, when surgical removal was combined with radium treatment, cure resulted.

EDITOR'S ABSTRACT.

TREATMENT OF SEBORRHEA OF AUDITORY CANAL AND OF CHRONIC ECZEMA OF EAR WITH THORIUM-X. I. HERZFELD, Monatschr. f. Ohrenh. **70**:1325 (Nov.) 1936.

Herzfeld uses a solution of thorium-X which emits alpha rays that are readily absorbed but act only on the surface. He applies the solution by rubbing it into the wall of the meatus several times in succession. All scales, crusts and secretion should, of course, have been removed previously. After two days the treatment is repeated. Especial attention should be given to the meatus acusticus externus.

EDITOR'S ABSTRACT.

ENLARGEMENT OF CIRCUMFERENCE OF NECK AS SYMPTOM OF LABYRINTHINE FISTULA. T. HASEGAWA and G. KONISHI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **41**:164, 1936.

Hasegawa and Konishi describe a new apparatus, the so-called cervicometer, which permits the registration of changes in the circumference of the neck. They point out that labyrinthine fistula is characterized not only by the well known nystagmus but by an enlargement of the cervical circumference. The cervicometer reveals an increase in the circumference of the neck after caloric stimulation of the labyrinth. In patients with labyrinthine fistula the caloric stimulation of the healthy ear produces a greater increase in the cervical circumference than does the caloric stimulation of the diseased ear. The authors assume that the enlargement of the circumference of the neck has its cause presumably in a shortening of the cervical muscles.

EDITOR'S ABSTRACT.

Larynx

LARYNGEAL TUBERCULOSIS IN CHILDREN. O. TARDY, Časop. lék. česk. **74**:1254 (Nov. 8) 1935.

Reviewing the literature, Tardy cites Pochitonov, of Leningrad, Russia, with a series of 5,000 children observed in a hospital for pulmonary diseases. Of these, 2,060 had tuberculosis, distributed as follows: chronic tuberculous intoxication,

38.8 per cent; tuberculosis of the bronchial glands, 31 per cent; pulmonary tuberculosis, 21.1 per cent; tuberculosis of the cervical glands, 6.2 per cent; tuberculous pleuritis, 2.25 per cent; tuberculous meningitis, 0.25 per cent; peritonitis, 0.25 per cent; lupus, 0.1 per cent, and tuberculous laryngitis, 0.05 per cent (1 case). Frischman, of Moscow, Russia, reports that the pathologic material from St. Olga's Children's Hospital from 1887 to 1921 showed the spleen to be involved in 86 per cent of the cases, the liver in 73 per cent, the lungs in 57 per cent, the meninges in 56 per cent, the kidney in 46 per cent, the intestines in 24 per cent, the pleura in 16 per cent, the brain in 10 per cent, the mesenteric glands in 6 per cent and the peritoneum in 5 per cent. The heart and the glands of internal secretion are regarded as immune, and the larynx is not mentioned. Teyschel, of Vienna, Austria, presents figures on the incidence of a positive reaction to tuberculin in children as follows: of those from 1 to 2 years old, 9 per cent; in those from 3 to 4 years, 27 per cent; in those from 5 to 6 years, 51 per cent; in those from 7 to 10 years, 71 per cent; in those from 11 to 14 years, 94 per cent. There were 8 cases of tuberculosis of the larynx in children from 10 to 15 years old in the children's division of the hospital from 1921 to 1934. During this period there were 986 cases of laryngeal tuberculosis in adults, so that the incidence in children under 15 years of age was about 0.8 per cent.

STULIK, Chicago. [AM. J. DIS. CHILD.]

EMPHYSEMA CAUSED BY PARTIAL COMPRESSION OF THE BRONCHUS BY TUBERCULOUS LYMPH NODES. L. CSONT, Orvosi hetil. **81**:37 (Jan 9) 1937.

A 2 year old boy was admitted to the hospital with the diagnosis of spontaneous pneumothorax. The boy had been examined three months earlier and tuberculosis of the hilus found. Now the left side of the thorax was enlarged, and there was very slight breathing. On the roentgenogram the mediastinum showed a dislocation to the right side. On the left side of the right lung the diaphragm was pushed down; the lung seemed to be very light, but everywhere the fine shadows of the bronchi showed the lung to be enlarged because of emphysema. On the left side in the hilar region there were two lymph nodes of hazelnut size, which had not been there three months before. The diagnosis was emphysema of the left lung caused by partial compression of the left main bronchus. A roentgenogram taken three months later showed normal conditions, and the lymph nodes in the left hilar region were diminished.

GÖTTCHE, Budapest, Hungary. [AM. J. DIS. CHILD.]

Nose

HAIMAN CORRELATION OF VASOMOTOR SYMPTOMS ASSOCIATED WITH NASAL MANIFESTATIONS IN CHILDREN. J. A. HAIMAN, Arch. Pediat. **54**:236 (April) 1937.

Haiman lists a group of apparently unrelated symptoms which he claims are associated and constitute a vasomotor syndrome. He states that the syndrome is clearly defined and that if one will follow the various symptoms mentioned, the diagnostic features will present themselves vividly.

The following symptoms are mentioned: swollen nasal mucosa; cough and postnasal drip; frequent colds and sneezing; lacrimation; lack of tonicity and late afternoon fatigue; an emotional, high strung, tense manner; gastrointestinal manifestations, such as vomiting and pain simulating appendicitis; cutaneous manifestations, vesicular rashes and urticarial phenomena, and cardiac manifestations such as tachycardia.

These symptoms will be found in cases of vasomotor rhinitis and nasal sinusitis, in cases of croup and tracheobronchitis and very occasionally in cases of hay fever and asthma.

ORR, Buffalo. [AM. J. DIS CHILD.]

CHRONIC SINUSITIS WITH EARLY BRONCHIECTASIS. S. CAMPBELL, Proc. Roy. Soc. Med. **30**:708 (April) 1937.

A case in a boy of 11 years is reported. At the age of 5 years he had an attack of pneumonia; at 6 years, diphtheria and pneumonia, and at 7 years, whooping cough and measles. Tonsillectomy and bilateral antrostomy were performed when he was 8 years old. After that he had repeated attacks of bronchitis and for the last two years had persistently brought up large quantities of sputum the first thing in the morning and again in the evening. He became short of breath on exertion. When he was admitted to the hospital, coarse rales were heard at the bases of the lungs, and the fingers were clubbed. With postural drainage 50 cc. of sputum was produced daily. Roentgenograms showed increased markings at the bases of both lungs. Iodized poppyseed oil 40 per cent showed slight dilatation of the bronchi at the base of the right lung. Both maxillary sinuses were opaque. The report is accompanied by a reproduction of a roentgenogram of the chest taken following the introduction of iodized oil and of one of the skull.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

ENDONASAL IMMUNIZATION AGAINST DIPHTHERIA WITH NONCONCENTRATED DIPHTHERIA TOXOID. F. BLAŽEK, Časop. lék. česk. **74**:833 (Aug. 2) 1935.

Blažek developed immunity to diphtheria in 50 per cent of children by intranasal application of undiluted anatoxin. The antitoxin titer was increased in 66 per cent of 56 cases. The only reaction noted was a mild elevation of temperature in 30 per cent of the subjects. The method may be of value in secondary immunization of children who have had a previous massive reaction, but the antigenic value of the material will have to be increased for better results.

STULIK, Chicago. [AM. J. DIS. CHILD.]

CHANGES OF MUCOSA OF ACCESSORY SINUSES CAUSED BY RHEUMATIC INFLAMMATION OF BLOOD VESSELS. R. RÖSSLE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:193, 1936.

Rössle demonstrates that in the severe sepsis-like borderline forms of rheumatoid arteritis there occur, in addition to the well known localizations of vascular inflammation, extensive necrosis of the mucous membranes of the nose, the oral cavity, the middle ear and the respiratory passages. The necrotizing diphtheroid inflammation observed in this disease is due chiefly to vascular changes of the type of periarteritis nodosa, but in the regions that are less severely involved, there are numerous small granulomas of the structure of the rheumatic nodules in the submucosa, for instance, of the pneumatic cells of the mastoid process, of the frontal sinuses and of the tympanum. The question of whether the exceptional localization of these changes in the aforementioned membranes is the manifestation of their excretory function for toxins is asked, because other excretory organs, such as the kidney and the intestine, are likewise severely involved in this disease.

EDITOR'S ABSTRACT.

Miscellaneous

SPEECH AS AN EMERGENT SPECIFICITY. J. H. MUYSKENS, Am. J. Psychiat. **93**: 857 (Jan.) 1937.

This is a preliminary report on 171 institutionalized children under 13½ years of age, with the following deductions: Thirty-four per cent had a defect in speech of sufficient severity to require special training. The children as a group were markedly inferior physically and physiologically as compared with the normal population. Those in the group with the lower intelligence quotients showed a definitely greater deviation from the norm than those in the group with the higher intelligence quotients.

Of 125 children whose medical developmental histories, which were complete, were analyzed, 14 per cent showed systolic murmurs and 50 per cent had thyroid, pituitary or other glandular dysfunction. Over one third of the children had hypertrophied turbinates, though one half of these had had their tonsils removed.

JAHR, Omaha. [AM. J. DIS. CHILD.]

DRAINAGE OF CEREBROSPINAL FLUID IN TREATMENT OF ACUTE HEAD INJURIES. D. H. WERDEN, Arch. Surg. **34**:424 (March) 1937.

There has always been considerable difference of opinion regarding the use of spinal puncture and the indications for surgical intervention in the treatment of acute injuries of the head. Werden in this comprehensive article shows that drainage of cerebrospinal fluid in cases of acute injury of the head is not only without danger but is highly beneficial. It is assumed that the clinical observations all point to an increase of intracranial pressure as the outstanding finding in cases of cerebral concussion and to an excessive secretion of the cerebrospinal fluid as the principal factor causing this pressure. This justifies drainage of the cerebrospinal fluid. The value of spinal drainage in preoperative care and in determining when surgical intervention is indicated is thoroughly discussed. The contraindications for spinal drainage are discussed. In this study all the patients were treated with spinal drainage. It was concluded that there was definite therapeutic value with this method of treatment in a large percentage of cases of injury of the brain ranging from mild concussion to severe injury of the head.

KAISER, Rochester, N. Y. [AM. J. DIS. CHILD.]

A CLINICAL SYNDROME INDICATING CESSATION OF SURGICAL DRAINAGE FROM AN ABSCESS OF THE BRAIN. J. M. NIELSEN and CYRIL B. COURVILLE, Bull. Los Angeles Neurol. Soc. **2**:11 (March) 1937.

Nielsen and Courville state that as soon as an abscess of the brain is properly drained there should be relief from headache, increase in temperature and pulse rate, improvement in the mental state, cessation of vomiting and acceptance of food and water. They attribute the rise in temperature to disturbance of the inflammatory reaction in the walls of the abscess and the increase in the pulse rate to diminution in intracranial pressure and release of bacterial toxins. If drainage is inadequate, a train of symptoms results, which Nielsen and Courville divide into three stages: First, the patient suffers from headache, irritability and restlessness and sleeps poorly—symptoms which probably result from an inflammatory reaction about the abscess. In the second stage signs of pressure, such as anorexia, nausea, vomiting, drowsiness, incontinence, stupor and often convulsions, appear. During this period the temperature and the pulse rate fall below normal. If no relief is given by proper drainage, the third stage supervenes, in which the patient lapses into coma and dies of respiratory failure, rupture of the abscess or meningitis. The authors identify these three periods with the three stages in the development of an abscess of the brain as described by Macewen. Appearance of such symptoms after drainage of an abscess indicates either that the drainage has become obstructed or that there is another abscess.

MACKAY, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

PATHOLOGIC CHANGES IN MÉNIÈRE'S DISEASE. W. E. DANDY, J. A. M. A. **108**:931 (March 20) 1937.

In previous reports Dandy has shown that Ménière's and pseudo-Ménière's disease can be cured by section of the auditory nerve or of the vestibular branch alone. The number of operations has now reached 170 in 160 patients, bilateral sections being performed in 10 instances. There has been no death and, except for early facial palsy in 4 cases, no after-effects from the unilateral operation. Like the cure for trigeminal neuralgia, that for Ménière's disease has antedated

disclosure of the underlying cause. Since the disturbances do not in themselves produce death, pathologic material is scarce. The absence of necropsy material throws the burden of pathologic disclosures on conditions observed at operation. From this source a series of gross lesions in 8 cases is offered as the unequivocal cause of Ménière's disease. Only lesions of the sensory root of the auditory nerve (the vestibular division) can produce Ménière's disease, and consequently lesions in the semicircular canals, such as concretions, are not a cause. However, although Dandy does not believe that concretions in the cochlear aqueduct can cause recurring attacks of dizziness and loss of hearing, he is not able to make a categorical denial that it is possible. The effects of irritative or occlusive concretions in this channel are not yet known. It would be difficult, however, to understand how an occlusion in the ductus cochlearis could affect the semicircular canals, which, although connected through the ductus endolymphaticus, should be independent of any secondary effect from an obstruction. Concerning the theory of Portmann and Aboulker, that the effects of pressure are transferred from the cisterna lateralis through the ductus endolymphaticus, one can be positive. That pressure in the lateral cistern does not in any way affect the semicircular canals or the organ of Corti is easily shown by the absence of effect in the great number of cases in which tumor of the brain is known to produce an increase of pressure in the lateral cistern and in which there is never any pathologic effect on hearing or vestibular function. The theory is purely fanciful, without any anatomic background. In no single operative exposure of the lateral cisterns in this series has any abnormality been observed; certainly, there has been nothing suggestive of localized meningitis, which is an almost impossible conception.

EDITOR'S ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.]

NASOPHARYNGEAL SEPSIS IN 2,056 CASES OF MENTAL DISORDER. T. C. GRAVES, Brit. M. J. **1**:483 (March 6) 1937.

This paper summarizes briefly 2,056 cases of infection of the nose and the throat occurring in the patients of the Birmingham Mental Hospital during eight years. The sexes were about equally represented. Adenoids were removed in 129 instances, and tonsils, in 1,186. Diseased nasal sinuses were found in 1,425 patients. Graves says that mental symptoms may be among the first manifestations of "closed nasal sinus infection." The inference to be drawn is that certain psychoses may be improved or cured if infected sinuses are recognized and treated.

ECHOLS, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

TREATMENT OF ACUTE RHEUMATISM IN CHILDHOOD. R. LIGHTWOOD, Lancet **1**: 613 (March 13) 1937.

In view of the recognition of the three phases of acute rheumatism—infection of the throat, the latent period and the rheumatic attack—it is the clinicians' duty to make practical use of this knowlege. The first problem is to prevent infection of the throat in rheumatic children. To this end sufficient rooms should be provided so that every patient with tonsillitis in a group of rheumatic children can be separated from the other patients.

As far as treatment of tonsillitis and pharyngitis is concered, Lightwood places chief reliance on painting the tonsils with a solution of strong protein silver and giving potassium chlorate by mouth. Activity should not be resumed until the pulse rate and the sedimentation rate are normal.

Clinical experience suggests that a complete tonsillectomy lessens the number of infections of the throat and may, therefore, he expected to be a help in suitably picked cases. "Certainly we ought not to advise the removal of normal tonsils from any child, even a rheumatic one."

Every patient showing any evidence of rheumatic activity is nursed almost flat, with but one pillow, is allowed no active movement except of the hands and

is fed by a nurse, so that the work of the heart is reduced to a minimum. When all signs of rheumatic activity have passed, a gradual return to ordinary life is permitted.

Acetylsalicylic acid has not yet been proved clinically to be of definite prophylactic value in the latent period. Of the many drugs that have been used with the object of checking the rheumatic infection, none is of primary importance.

LANGMANN, New York. [AM. J. DIS. CHILD.]

DISCUSSION ON THE CHOICE AND TECHNIQUE OF ANAESTHETICS FOR NOSE AND THROAT OPERATIONS. T. B. LAYTON and others, Proc. Roy. Soc. Med. **30**:923 (May) 1937.

T. B. Layton believes that a local anesthetic is indicated in nasal operations and bronchoscopic work but that a general anesthetic is better for such operations as tonsillectomy, esophagoscopy and direct laryngoscopy. He prefers morphine and scopolamine as premedication for both local and general anesthesia.

H. E. G. Boyle is partial to basal anesthetics for pharyngeal operations, preferring pentobarbital sodium, avertin and paraldehyde in that order. He then uses gas-oxygen and as little ether as possible.

W. S. Thacker Neville reports his experiences with various types of preanesthetic agents and describes his procedure of operating in cases of nasopharyngeal disease. He has found the preoperative use of morphine and scopolamine, sodium soneryl (sodium butyl ethylbarbituric acid), evipal (C-C-cyclohexenyl-N-methyl-barbituric acid) and pentothal sodium (sodium ethyl 1-methyl butyl barbituric acid) the most useful.

A. W. Matthew believes that the long-acting basal narcotics, such as avertin, pentobarbital sodium and morphine should not be used as preoperative medication for nasal and pharyngeal operations, because they tend to impair the defensive reflexes. He discusses in some detail his methods of operating under local and general anesthesia.

D. F. A. Neilson considers general anesthesia more beneficial to the patient than local anesthesia. He believes premedication with morphine contraindicated for tonsillectomy with local anesthesia. He discusses the dangers of postoperative hemorrhage, the matter of position of the patient and other features of operative technic.

J. H. T. Challis considers omnopon (a mixture of opium alkaloids) and scopolamine the preoperative drugs of choice for operations on the upper air passages, with anesthesia by gas-oxygen and ether.

Z. Mennell thinks there is nothing so safe before operation, especially for children, as paraldehyde. He considers it dangerous to use a combination of chloroform and epinephrine.

Several other discussers give opinions on the use of both preoperative and operative anesthetics.

CAMPBELL, Philadelphia.

DETERMINATION OF DIRECTION OF SOUND AND VESTIBULAR APPARATUS. A. GÜTTICH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **142**:139, 1936.

Güttich demonstrates that the detection of the direction in which a sound originates is made possible chiefly by the essential portion of the auditory organ, the cochlea. However, when the eyes are automatically focused in the direction of the sound, the cervical reflex and the reaction to stimulation of the semicircular canal are of great significance.

EDITOR'S ABSTRACT.

DISCOVERY OF CALORIC NYSTAGMUS. M. SUGÁR, Monatschr. f. Ohrenh. **70**:1328 (Nov.) 1936.

Sugár objects to a report which Rejtö published in several recent otolaryngologic publications, giving Bárány credit for the discovery of caloric nystagmus.

The author shows that caloric nystagmus had been described by other otolaryngologists before Bárány's report appeared and that Bárány's experiments on human subjects were based exclusively on animal experiments made by Högyes, but that Bárány never even mentioned Högyes. The author says further that the Vienna Academic Senate accused Bárány of obscuring the merits of others. In conclusion he points out that, although it cannot be denied that Bárány had certain talents, he should not be overestimated, for his discovery of caloric nystagmus was based on Högyes' animal experiments; his pointing test was developed on the basis of Graefe's touch test; his noise drum is not an indispensable instrument, and his suggested surgical treatment of otosclerosis was a failure.

EDITOR'S ABSTRACT.

DISCOVERY OF CALORIC NYSTAGMUS. A. REJTÖ, Monatschr. f. Ohrenh. **70**:1335 (Nov.) 1936.

Rejtö, in replying to Sugár's charge that Bárány failed to give credit to Högyes' experiments, says that in a summary report Bárány did mention a number of papers published by Högyes and that consequently it is erroneous to say that Bárány entirely neglected to mention Högyes.

EDITOR'S ABSTRACT.

Book Reviews

Practical Otology, Rhinology and Laryngology. By ADAM EDWARD SCHLANSER, M.D. Price, $4.50. Pp. 315, with 81 illustrations. Philadelphia: Lea & Febiger, 1938.

This book was written for the purpose of affording a reference and guide book for army medical officers during their routine duties in otorhinolaryngologic practice in peace time service. The author has succeeded admirably in his purpose.

The usual textbook setup has been changed to conform to the cases which present themselves to the medical officer. Thus the outline stresses the many complaints and symptoms, and the reader will observe that the text simplifies all of these, to the end that the medical officer assigned to the otorhinolaryngologic service will be given full instructions in the manner of handling these cases more efficiently.

Theories and controversial subjects have been studiously avoided, the entire book stressing practical clinical data. Anatomy and physiology have been entirely omitted, and the pathology limited to the barest essentials, in order that the reader may quickly comprehend the clinical evaluation without the confusion which so often arises when one seeks important facts in a great hurry.

In evaluation of the entire text, it is evident that the author has presented a sound practical otolaryngologic theme, with logical conclusions and safe procedures. The literary style is excellent and the instructions lucid and to the point, enhancing the value of the book.

As stated in the preface, this book presupposes a grounded knowledge of the fundamentals of otolaryngology; hence the omission of the anatomy, physiology, fuller pathology and kindred basic principles. For this reason and because of the entire arrangement, the value of this good text will necessarily be limited to medical officers in particular, though this reviewer cannot help feeling that specialists will find this book valuable as collateral reading.

The graduate student in otolaryngology as well as the resident in this specialty will find the work useful for review purposes. The hospital intern may often find some valuable data, even though he is not grounded in the fundamentals thereof. In fine, it can be said that, though the work has been presented chiefly for medical officers on military duty, it should find a great range of usefulness as collateral reading for otolaryngologists and for those training for this specialty.

WILLIAM GORDON, M.D., Philadelphia.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.
Place: Brussels, Belgium. Time: September 1939.

Hungarian Otolaryngological Society

President: Dr. J. Treer, Bocskay-u. 9, Szeged.
Secretary: Dr. G. Kelemen, Reáltanoda-u. 9, Budapest.

Sociedad Rioplatense de Oto-Rhino-Laringología (Argentine Section)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: St. Louis. Time: May 15-19, 1939.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Washington, D. C. Time: Oct. 9-14, 1938.

American Bronchoscopic Society

President: Dr. John D. Kernan Jr., 120 E. 75th St., New York.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.

American Laryngological Association

President: Dr. George B. Wood, 504 Physicians' Bldg., Philadelphia.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Frank E. Kittredge, Masonic Temple, Nashua, N. H.
Southern.—Chairman: Dr. Francis E. LeJeune, Maison Blanche Bldg., New Orleans.
Middle.—Chairman: Dr. T. R. Gittins, Davidson Bldg., Sioux City, Iowa.
Western.—Chairman: Dr. Frederick G. Sprowl, Medical Arts Bldg., Spokane, Wash.

American Otological Society

President: Dr. Isidore Friesner, 36 E. 73d St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

---

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.